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OF THE

EPIDEMIOLOGICAL SOCIETY

OF

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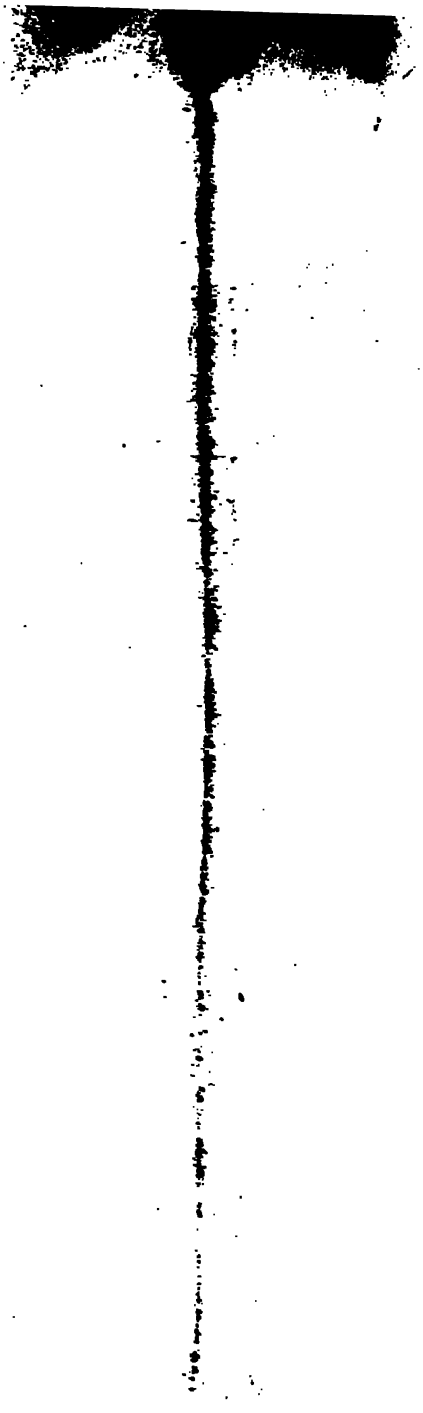

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TRANSACTIONS

OF THE

EPIDEMIOLOGICAL SOCIETY OF LONDON.

A SKETCH OF THE RECENT CHOLERA EPIDEMIC IN SYRIA.

By JOHN WORTABET, M.D.,

Physician to St. John's Hospital, Beyrout.

(*Read: December 8th*, 1875.)

[Abstract.]

Early in the spring of this year a few cases of Asiatic cholera were reported to have occurred in Hamah, 35 deg. lat. N., long. 37 deg. E.—a town of 40,000 inhabitants, situated on the banks of the Orontes, and lying on the high road between Aleppo and Damascus. How the disease should have appeared in so inland a place without its being known to exist in any part of Turkey or any of the provinces contiguous to it was a mystery, and the first reports were discredited. But as the news gained strength every day, the government ordered Dr. Sestalozza, the sanitary officer of Beyrout, to proceed to that place, inquire into the facts of the case, and report to Constantinople. This gentleman, who is highly qualified and experienced, confirmed the statement that the disease was really there, though he could not tell how it was originated. Two bodies of troops were said to have come there shortly before the disease had appeared—one from Arabia and another from Anatolia—but it was not known that cholera existed in either of those provinces, or that they had left any traces of it in their long march. Dr. Sestalozza was inclined therefore to believe that it must have arisen from some old germ which had become active under conditions favourable to its development, the presumed conditions being the situation of Hamah on the two sides of a river, the extremely insanitary state of the town, and unknown climatic influences. If this view could be sustained, it would be highly interesting in supporting the opinion of some that disease germs may be dormant for an indefinite length of time, and then break out into an active

and rapid state of development. But obscure to set aside the commonly re cholera always travels with human beir soiled with choleraic dejecta, and that th cannot be maintained for a very long ti reason to believe, though sufficient evide the troops from Yemen, in Arabia, brong them. It is certain that they came in t condition; that they had typhoid fever a was very fatal; and that cholera broke o in Hamah, and then on the same side town where their barracks lay. The int rities to conceal any fact connected with tion of infected troops adds to the prol rather brought by them into Syria than broken out from some germ which hac since the last epidemic in 1865.

Cholera appeared in Hamah about the continued till July, and is said to have number of victims. The intercommunica town and other places in Syria was left weeks the disease spread into the neighb towns, and in the latter part of July an more or less in Aleppo, Damascus, Beyro Latakia, and a number of the other mi brought into Damascus by a battalion of and continued there from June 13 to August. The number of deaths from c variously estimated from 5,000 to 1 consul had all the fresh graves coun ducted the corresponding average de obtained the following results:—M Christians, 278; Jews, 147—total 9, among the troops. The small ratio of deaths arises from the fact that a la taken refuge in the neighbouring mo of the epidemic.

While the epidemic was raging in bers of the Jewish and Christian part of the Moslems flocked to Moun other places. The communication fumigatory process was afterwards between Damascus and Beyrout, w useless. Nor were any subsequen carried out between inland places, tive of good results.

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The second case was that of Beyrout, who lived [illegible] from the place where the [illegible] slight discrepancy as to the [illegible] quarantine returns of [illegible] on the 25th of July, and [illegible] 27th. The infection [illegible] brought into the Jewish [illegible] by a fugitive family from [illegible] not far from her own. No one of had cholera, nor is it known that a Another Jewish family from Dama soon after, and on the 2nd of Au in the same quarter was attacked of the 3rd. She lived in a low and just above was a Jewish fami wife, and a number of children. of six years, was taken ill with death, and at the same [illegible] from the place was also [illegible] morning of the 4th [illegible] covered. As I [illegible] quarter cases [illegible] exception of [illegible] four cases [illegible] ise of [illegible] inhabitants [illegible] appearance of [illegible]

and rapid state of development. But the data are too obscure to set aside the commonly received theory that cholera always travels with human beings, or with articles soiled with choleraic dejecta, and that the life of such germs cannot be maintained for a very long time. There is some reason to believe, though sufficient evidence is wanting, that the troops from Yemen, in Arabia, brought the disease with them. It is certain that they came in the most insanitary condition; that they had typhoid fever among them, which was very fatal; and that cholera broke out first among them in Hamah, and then on the same side of the river in that town where their barracks lay. The interest of the authorities to conceal any fact connected with such a transportation of infected troops adds to the probability that it was rather brought by them into Syria than that it should have broken out from some germ which had kept its vitality since the last epidemic in 1865.

Cholera appeared in Hamah about the end of March, and continued till July, and is said to have carried off a large number of victims. The intercommunication between that town and other places in Syria was left free, and in a few weeks the disease spread into the neighbouring villages and towns, and in the latter part of July and August prevailed more or less in Aleppo, Damascus, Beyrout, Tripoli, Antioch, Latakia, and a number of the other minor places. It was brought into Damascus by a battalion of troops from Hamah, and continued there from June 13 to about the middle of August. The number of deaths from cholera in that city is variously estimated from 5,000 to 10,000. The French consul had all the fresh graves counted, and, having deducted the corresponding average deaths in other years, obtained the following results:—Mahommedans, 8,894; Christians, 278; Jews, 147—total 9,319, including deaths among the troops. The small ratio of Christian and Jewish deaths arises from the fact that a large part of them had taken refuge in the neighbouring mountains on the outbreak of the epidemic.

While the epidemic was raging in Damascus, large numbers of the Jewish and Christian population and a small part of the Moslems flocked to Mount Lebanon, Beyrout, and other places. The communication was at first free, but a fumigatory process was afterwards established in a station between Damascus and Beyrout, which of course was wholly useless. Nor were any subsequent measures of this kind, carried out between inland places, either efficient or productive of good results.

rhœa. He died on the 28th. The other was a young Moslem girl, who made a very slow recovery, but it is quite unknown how she got the disease farther than that people from Damascus were daily coming into Beyrout and living in different parts of the town.

After these first few cases the disease broke out at so many different points that it was impossible to trace it or follow out its history and progress in a connected manner. There was, however, one common feature to these different points at which it appeared, namely, the tendency of the disease to spread from one infected individual to others around him. This fact has been so often noticed during the present and past epidemics that it has given rise to the popular notion, held by a few scientific men, that cholera is a highly contagious disease.

The water supply of Beyrout is such that we cannot believe that it was through this medium that the disease was communicated. Up to this summer the town was supplied from deep wells containing spring water. The wells are dug for a short distance through soft earth, then through stratified rock (hard limestone), and the water is not generally reached at a less depth than 100 feet. It is hardly possible therefore that the dejecta of cholera patients should percolate through such a material and depth so as to contaminate the water, and produce so speedy results as those mentioned above. Of running water there is but one spring, whose water is conveyed in a conduit to a short distance, and its use is confined to a very small part of the town where there were but few cases of cholera. Recently, the water of the Dog river (the ancient Lycus), a few miles to the north, was brought into the town by an English company, and the inhabitants have just begun to use it. It is conveyed in cast iron pipes, but its use has not become universal, nor could the disease in any way be traced to it.

During the present epidemic more than 50,000 people left Damascus and Beyrout and went up to the Lebanon villages. Some of them doubtless carried the infection with them, and a few cases of cholera occurred among them; but of the many villages to which the refugees went I heard of only three places where the disease showed any tendency to spread. As this immunity of the mountain heights from the ravages of the epidemic is a highly interesting fact, and has always mitigated the scourge in Syria by affording a safe retreat to the inhabitants of the towns and plains, it may be useful to mention three limited outbreaks in the villages of the Lebanon.

Sook-el-Gharb, a village nine miles south-east from Beyrout, built on a rocky soil, about 2,500 feet above the level of the sea, is a favourite resort of some people for escaping the summer heat of the town, and this year was far too crowded with fugitives from the epidemic. I have been credibly informed that one house, which has only a few rooms, was occupied by no less than thirty-nine persons; and every other house in the village was more or less densely inhabited. A quarantine had been established in Beyrout and Damascus, but it is known to have been often broken secretly; and a young man who spent the night out of the village was taken ill with cholera on the following day (August 9) and died of it. The disease appeared soon after in two others, and on the 12th three more were attacked. Of the six thus attacked three died, but in less than ten days from the date of the young man's death all traces of the disease in the village had disappeared. In Hammana, a village higher up on the mountains, and lying directly to the east from Beyrout, there was an outbreak of the disease about the 10th of August, imported probably by muleteers from Beyrout or Damascus. Eleven persons were said to have been attacked in the space of a few days, of whom four died. I saw the last case on the 21st. He recovered, and it is not known that there was any other case after him. The same short history is true of Sughbin—a high village on the eastern slope of the Lebanon, facing the plain of Cœle-Syria, where a number were attacked and a few died. From none of those places did cholera spread to neighbouring parts, though Mount Lebanon is thickly studded with villages, and in the case of Sook-el-Gharb there are no less than six of them at different distances within the radius of a mile from that place.

It would thus appear, from the above-mentioned cases, and from what is known of former epidemics, especially that of 1865, the last which appeared in Beyrout, that mountain heights are altogether unfavourable to the propagation of cholera. For when it is imported there from low plains it does not show a disposition to spread, nor does it continue more than a few days. Whether the cause be the simple matter of height, or the presence of a large amount of ozone, or the purer state of mountain air, or the character of the soil and the ground water, it is difficult to say; but the fact seems to be well established, and the adverse agent appears to be most probably of an atmospheric nature.

A practical conclusion which may be safely drawn from this fact is the inutility of imposing quarantine during an

epidemic of cholera in Syria, between the plains and the mountains. Not only are the villages dependent on the towns for their material supplies, and on this ground alone it is impossible to stop the communication, but their elevated position forms a sufficient barricade against the invasion of the disease. An attempt, however, was made during the present epidemic to keep a quarantine in some of the villages, and in no place was it so rigidly enforced as in Sook-el-Gharb. When the slight outbreak appeared there, all the quarantines were given up on the mountains without any injurious results.

The epidemic in Beyrout was comparatively mild, and not of long duration. It began in the latter part of July, and the last fatal case was on the 15th September. There were 8 cases in July, 51 in the first week of August, 44 in the second, 28 in the third, 7 in the fourth, and 11 in the first half of September. Of these cases of death there were 79 males and 70 females; of Mahommedans 78, Christians 68, Jews 7. Their ages, as far as the quarantine returns can be relied on, and they are only approximately true, were as follows:—

From 1 to 10 years	29
„ 10 „ 15 „	10
„ 15 „ 20 „	12
„ 20 „ 30 „	25
„ 30 „ 40 „	20
„ 40 „ 50 „	23
„ 50 „ 60 „	14
„ 60 „ 80 „	9
Ages known	142

On the same authority the number of fatal cases was 153, and that of attacks was 274. This last number cannot be altogether true, because it is extremely difficult to find out in a large town, not under strict medical surveillance, every case of incipient cholera, and to distinguish it from ordinary diarrhœa. For I have been led during the present epidemic to doubt the accuracy of the rule given by some authors, that during the prevalence of cholera every case of diarrhœa is to be considered as a genuine instance of the disease. That it should be so considered practically, from prudential motives, is certainly the safest course; but where it becomes a question of relation between the number of attacks and the number of deaths the case is quite different, though it must be admitted that simple diarrhœa and that which precedes the graver symptoms of cholera cannot always be distinguished from each other. And yet I have often ob-

served simple cases of diarrhœa—sometimes neglected and protracted—which did not terminate in a clear development of cholera; so that it may be possible, during an epidemic of this disease, that there may be two different classes of diarrhœa—one simple and the other specifically choleraic. From the 17th of July to August 31st there were 159 deaths from other diseases; but I have no means of knowing how this number compares with the mortality of corresponding months of other years, nor am I sure that it does not include some of those obscure masked cases of cholera which are easily confounded with other diseases.

The population of Beyrout is generally estimated at 60,000 to 80,000 inhabitants, and it is supposed that about two-thirds of this number left the town for the mountains on the appearance of the epidemic. Taking, as a rough guess, the population left in the town to be about 30,000, and the cases of death from cholera 150, the ratio will be 5 per 1000. The flight to the mountains was probably beneficial in two ways: it removed a large part of the inhabitants from the risk of infection, and left the remaining part so thinly distributed that the pestilence lost one of its most favourable conditions for extensive propagation. Nor is Beyrout, though a seaport, a favourable place for the fullest sway of cholera. The old town is not too thickly inhabited, and the houses which lie outside of it, and which form by far the largest part of the city, are more or less separated from each other, so as to give free vent for currents of air between them. The sewerage of the old town is led in a conduit, defective in some places, to the sea; the waterclosets in the houses outside empty themselves into deep pits in the earth, and are cleaned out from time to time. The use of disinfectants was carried out more or less efficiently in every case which came under my care; and I recollect a number of instances in which the free use of sulphate of iron seemed to check the further progress of the disease in the infected spot. To these causes for the mildness of the epidemic in Beyrout may be added that it was not very severe throughout Syria, relatively to former visitations; and it was possibly for this reason that it moved so slowly from place to place—appearing in Damascus more than two months after it had declared itself in Hamah; and in Beyrout six weeks after it had been in Damascus, though the intercommunication between all inland places was never seriously interrupted.

The means which the authorities employed for limiting the extension of the disease were the daily cleaning of the streets, the prevention of the sale of unripe or decomposed fruits

and vegetables, and the employment of medical men in the Quarantine and Municipal departments for investigating and treating cholera cases. Some stamped papers were distributed among the medical men of the town for prescribing gratis to needy patients, and 4,000 piastres were given to the Prussian Sisters and the Sisters of Charity for feeding the poor, and this sum was considerably augmented by private contributions. Disinfection was understood to be enjoined in every case, but the execution of this important measure depended much on the conscientiousness and influence of the physicians.

The summer was unusually cold, owing to a nearly continuous succession of south and south-west breezes. The mean maximum of the temperature during the month of August was 88·5 deg. F., and the mean minimum was 72 deg. F. There were very few cases of ophthalmia and dysentery, which are commonly so rife during the summer months—specially the former—a few intermittent fevers, an occasional case of insolation, a special kind of febricula, which is complicated with gastric symptoms and accompanied by severe pains in the loins and knees (and hence called by the natives the *knee-fever*), and towards the end of the epidemic a few cases of malignant remittent fever.

It may be useful to compare the epidemic of this year (1875) with the last one which visited Beyrout (1865), and which began on the 1st of July, and was not through before the middle of September. It took away 579 victims (July 161, August 390, Sept. 28)—nearly four times as many as it did this year—of whom 268 were males and 311 females. [See Dr. Lorange in the *Berliner Klinische Wochenschrift*, 6th Nov. 1865.]

NOTES ON THE RECENT HISTORY OF THE PLAGUE IN THE PROVINCE OF BAGHDAD.

By WILLIAM HENRY COLVILL, Surg.-Major,
Civil Surgeon, Baghdad.

(*Read June 13th*, 1877.)

Writing of plague in and around Baghdad at present is very much like reviewing a tragedy of which only two acts have appeared, and no one knows the end. In order to make my account better understood, I would premise some observations on the general features of the country, after that glance at the towns, and then give some account of the domestic life of the people, who are little understood even by Europeans who have dwelt for years in their midst.

Mesopotamia, as is well known, is entirely alluvial soil, bounded on the south by the desert of Arabia, which appears in ridges of sandstone and limestone like the waves of the Atlantic, and on the north by the same sandstone and limestone, forming the table land of Persia. Mesopotamia was in fact a portion of the Persian Gulf, and as deposits were thrown down, certain bays and deltas were left, forming at the present day the three principal marshes. The chief of these is the sea of Nejef, originally called the Chaldean Lake, with the Hindeah Canal which leads into it. That this is so is obvious from the fact that this process of silting is still going on in the harbour or bay of Koweyt at the top of the Persian Gulf, where at no distant period (geologically speaking) a lake will be formed similar to the Chaldean Lake.

An arm of the sea, a creek, with four fathoms of water, leads up to a place called Bunder Zobeir, within eleven miles of Busreh; and any one standing on Bunder Zobeir can trace the outline of a canal which once existed and opened into the combined Euphrates and Tigris. This canal, with the creek, would represent the Hindeah Canal; the bay of Koweyt, thirty miles long and fifteen broad, would represent the sea of Nejef, and though at first the surplus water will fall directly into the Persian Gulf, eventually it will enter into the main drain, *i.e.*, the combined Tigris and Euphrates,

and will therefore represent the Shatt-el-Shinafeah which carries the surplus water of the sea of Nejef into the Euphrates. It must be remembered that the Hindeah Canal is no modern affair as is generally supposed; for more than 3,000 years ago a tribe of Arabs called Hindanee lived on its bank and paid tribute to the King of Assyria. Another proof of its antiquity is to be found in the fact that the ruins of Babylon would be submerged if the Hindeah Canal did not carry off half the water of the Euphrates; so that the canal must have existed from the time of Babylon at least.

We next come to the Affiche Marsh, which is fed with water by the Danarah Canal. Some doubts might exist as to whether this was a swampy hollow left at the time when the surrounding country was formed, but at any rate it is a marsh which has existed as long as the most ancient cities we know of; for here on its border are the ruins of Nipur; while on the river carrying off the surplus water of this marsh into the Euphrates, are the ruins of the city of Warkah or Erech. The next great marsh which I want to mention is much more easily traceable to a hollow left in the formation of the ground, because it is nearer the Persian Gulf and much less filled up than the Affiche Marsh. This marsh leads from the left bank of the Tigris, at a point two-thirds from Baghdad and one third from Busreh; it carries off half the water of the Tigris and branches out, dividing and sub-dividing, to the foot of the Persian Hills, and to a place marked on the Map as Howiza, in the direction of Shuster, and the surplus water, after the deposition of the *débris*, falls into the combined Tigris and Euphrates. One other, I do not know whether to call it a canal or river (the Arabs call it the river Hai), passes diagonally from the Tigris into the Euphrates, and in spring overflows its banks in many places. Whether this is natural or artificial I am unable to say, but it is a curious fact that while on its banks are the remains of the most ancient cities we have heard of (and they are numerous), the banks of the Tigris, from the point where this canal leaves it to the Persian Gulf, are destitute of ruins. It would almost seem as if this were the original bed of the Tigris to its junction with the Euphrates. The minor marshes are those formed for the purposes of cultivation, the water being let on to the ground through canals which an Arab horse can either leap over or wade through. These can hardly be called swamps, since their banks are all cultivated. There are also one or two marshes, made, either by the overflowing of the rivers at their height, as down in the neighbourhood of Busreh, or by the burst-

ing of ancient canals, as in the case of the marsh to the west of Baghdad, where the trench of Artaxerxes let the water on to the plain. But these marshes have evidently been in existence from very early times, as for instance this one to the west of Baghdad; because one sees where the canals from the Euphrates end a large fortress once existed, now called the ruins of Akar Keef, and long supposed to be the tower of Babel. This fortress protected the entrance into Babylonia, which lay between the canals from the Euphrates and this marsh to the west of Baghdad. Of the two rivers which drain this land, the Euphrates sends half of its waters through the Hindeah canal into the sea of Nejef, a lake sixty miles long by thirty broad, where all its silt is deposited. Almost a quarter of the water of the Euphrates enters the Damarah Canal, and deposits its silt in the Affiche marsh, which is twenty-two miles long and ten broad. In like manner half the water of the Tigris goes to the Persian frontier and deposits its silt in the Howiza marsh, which is of great extent. But these ought not to be called marshes at all, they ought to be designated lakes.

The rainfall in this province varies from eighteen to twenty-five inches, and falls chiefly in December, January and February. The winds from the south bear clouds from the Persian Gulf and carry them, often without a drop of rain on the low ground, to the Persian hills, where the moisture is deposited as snow, and it is often when the wind blows from the north, bringing back a portion of the clouds, that the heaviest rains occur in the low countries. These hills are at the sources of the Tigris and Euphrates, and on the quantity of snow therefore depends the inundation of the rivers. But any great fall of snow is accompanied by a correspondingly large fall of rain in the plains; and to an unusual fall of rain, more than to inundations, increased sickness is probably due.

The saying is, and it is certainly true for miasmatic fevers, "the hotter the season the healthier it is"; yet Europeans have come to me telling me that their family physicians had recommended them to come out to Baghdad because they had delicate lungs. A more deadly mistake could scarcely be made, for the extreme heat here is even more injurious to a consumptive patient than extreme cold. The reason is obvious. In the cold, the patient can be wrapt up for exercise, and his room kept at an agreeable temperature; while in the extreme heat there is not only great languor and physical depression, from the rarity of the atmosphere, but the exercise, which can be taken only in the early morning,

exhausts an invalid instead of invigorating him; as he sleeps at night too, which he must do on the housetop, with very little covering, he perspires, then comes a breath of air and he catches a chill, and this will happen two or three times in the night, so that here in the hot weather a real galloping consumption can be observed.

It seems almost absurd to say anything of the vegetation of a province which does not possess a forest tree; where the only brushwood is a thin fringe, either tamarisk or dwarf poplar, on the margin of the river and sprinkled about some of the ancient canals. The plants, such as they are, are European in their character, and a marsh here is composed of water and bulrushes; the rushes grow sometimes to the height of 15 ft., but near towns they are too valuable for building purposes, for matting, and for fuel, to be allowed to rot in the swamps. Date trees of course there are, chiefly at Busreh, where they are watered by the tide, a few round the towns, and a sprinkling almost all the way along the Euphrates from Hillah to its junction with the Tigris; but as those on the Euphrates are all past their prime, and no new ones have been planted, the banks of the Euphrates will be as bare as those of the Tigris.

The towns are not numerous, and can bear being clearly divided into three classes: ancient, modern, and Mahommedan. The ancient towns are Baghdad, Hillah, and Busreh. Baghdad, on the high way from Babylon to Ecbatana, was an important city when it was taken 1300 B.C. by Tugultininiss, first of the name, King of Assyria, when on his way to conquer Babylon. The massive, cuneiform inscribed brickwork in the bed of the river, shows that it must have been an important place. The town of Hillah I consider for my present purpose ancient, because it is built out of the ruins of Babylon, and rests on its site. Busreh would appear to have had its site shifted from more inland; still, though no cuneiform inscriptions are found thereabout, it must be considered old. The modern towns are Amarah on the Tigris, Nassreah on the Euphrates, where it joins with the river Hai, and Rumoetha higher up, also on the Euphrates. Amarah is situated in the fork where the waters of the Tigris go off to the Hawiza marsh. Thirteen years ago, when the Arabs there were in revolt, troops were sent down to this then deserted spot; traders followed; and now Amarah is a brick town of about 5,000 inhabitants on a clean unpolluted site. Nassreah, with 3,300 inhabitants, was built six years ago by the chief of the Montafik, for no other reason than that he wanted to make for himself a name. Its site,

though unpolluted by any former city, is very low. Rumoetha is a small well-built town of about 300 houses. It sprang up twenty-five years ago, when some other towns on the Euphrates were deserted. The towns of the Mahommedan period are Nejaf and Kerbellah, the Holy of Holies of the Shea faith, where the bodies of Sheas who can afford it are sent for burial, under the belief that, having been buried in those sacred places, at the last day they will escape to heaven without a final examination. Strange to say, the graveyards of those places are farmed to a Jew, and Jewish watchmen nightly guard the burial ground to prevent surreptitious interment. But from the mass of bodies buried within so small a space, the state of those towns is beyond belief. So bad is Kerbellah, that if you look at the surface of the wells you see a film of fat which is simply human oil; and it has been told me, and I believe it though I have not verified it, that whoever sits on the ground or on a sun-dried brick sufficiently long, has his clothes stained with grease. One little point, which is creating a discussion in England, as it is connected with this subject might be mentioned here: it is that "coffin" is a purely Arabic word, and does not mean any box in which the dead body may be enclosed, but simply the cloth in which the body is wrapt. All the bodies at Kerbellah and Nejaf are buried in wooden cases which are called merely boxes in Arabic, while the term coffin is confined to the winding sheet simply. Traders here import coffin cloth, the flimsiest of its kind, laden with starch and pipeclay, which they sell especially for winding sheets, which alone are used in other towns; it is only at Kerbellah and Nijef that the bodies are buried in boxes, because most of them are brought from great distances. There are two other towns, Samaweh and Divanyeh, both on the Euphrates, but I know nothing of their history, though I suppose at some former modern time they were outposts for troops, and rose to be small towns. In none of these towns is such a thing as a drain known, and the earth underneath must do the whole work of disinfection, slightly aided by fresh dust blown in from the desert. That might be well enough for a time if the soil were left alone; but as new buildings are continually being erected to replace the wretchedly built houses, either the soil is dug up and thrown into the streets, or the streets themselves are dug up, according as the owner wishes to elevate or depress the site, thus exposing noxious matter to the influence of the atmosphere. It may yet be well remembered how exceedingly unhealthy we made Bushire for years after our expedi-

tionary force was there, simply by our energetic attempts at cleaning the town, and disturbing the filth of ages. There is no remedy that I can see for this state of things, unless the impossible supposition, that the whole populations of the towns be shifted; and this only shows us what a benefit the conquerors of old in this part of the world unwittingly conferred upon their subjects when they moved their capitals to fresh sites.

As there can be said to be only two classes of people in these towns—the middle and the lower—their houses are divided into two kinds, though the plan is actually the same for both. The better class are built upon brick arches, which form cellars called "serdabs", in which the families live during the great heat, and have verandahs in front of them; while the poorer houses have neither of these, and the people live immediately in contact with the ground. Every house, however large or small, is furnished with a courtyard, which in the better classed houses is bricked and has bottle-shaped holes for the reception of refuse, while in the poorer houses the refuse is absorbed immediately by the ground.

Having now described the towns, I would say something about the villages. A certain number of villages, larger than others, are built of mud and sun-dried bricks, such as Tanaby, the capital of the Hindeah. In these live petty governors who rule a circle of villages, the radius being about fifteen miles. The huts in these smaller villages are either of mud, of red matting, or of tents which are covered in with cut brushwood in the winter. Now these villages or groups of huts are all occupied by cultivators of the soil, and they can be distinctly divided into four classes: one class being the villages on high ground, where the irrigation takes place with skin buckets on account of the height of the bank, and the ground being dry, every advantage is received from the antiseptic influence of the earth. In the second class the villagers spend only the season of cultivation on the ground, and return to the towns or larger villages for the rest of the year. In the third class the villagers are driven, on account of the rivers inundating the country, from their rice-fields, and forced to move their habitation twice a year, whereby the ground they occupy during the greater part of the year is thoroughly cleansed. In the fourth class the villages are permanent, and built on ground which is a foot or two lower than the surface of the water in spring. At this season the ground is so saturated with moisture, that the refuse of these villages, which takes sometimes a greenish colour, and is sometimes dark blue oily

fluid, can neither be absorbed nor evaporated, for all the moisture it loses it regains from the ground. This oily fluid is everywhere in the path, so much so that bricks have to be placed as stepping stones between the huts. It soaks up and stains the matting of which the hut is chiefly made; in fact the filth is simply indescribable. The local authorities are not to be blamed, because for some reason or another they are frequently changed.

The population of this province may be roughly taken at a million, giving about twelve persons to the square mile. It can be divided into the inhabitants of towns, the inhabitants of villages, who are semi-settled Arabs, and the Bedouins, with whom at present we have nothing to do. The semi-settled Arabs who live in the villages, though they may have little ready money, are not to be considered very poor.

I desire to give some account of the domestic life of the inhabitants of towns, because so little is known of it, and the general impression is that the people here are only a set of dirty natives. In doing so I have adhered to the two great divisions of Jews and Mahommedans, leaving out Christians, who are too few and divided to require notice. In Baghdad alone the Jews are about 20,000 in number. The houses of the rich are large, and their poor are better sheltered than our own. But, though they follow with servility the laws of Moses, their houses are the dirtiest in the province. The furnishings of the rooms, however wealthy the occupant, are old and uncleaned, and the carpets ragged and unswept. Little attention is paid to the cleanliness either of the cooking utensils or the table service. The bedding is seldom clean, and never aired. The occupants, though the houses are large, sleep wearing the same clothes by night as by day, huddled up and locked into one room for safety. They obey the law by dipping at given times into a well of brackish water, while they greatly neglect the bath of soap and hot water. Sheep and fowls are subjected to examination by the priest. Not so fish, however, which with the oil of sesamum (*sesamum Indicum*), enters largely into the food of both rich and poor, and it is no exaggeration to say that on a hot day one can easily smell a Jew. Drink, not being forbidden, is taken in moderation, while the rabbis fine heavily all found drunk. The Jews have no slaves, but engage their own people in domestic service. The servants are always fed and often clad, and get wages from year to year, but the money is allowed to remain with the master at interest, till the sum accumulated is sufficient to make the man a shopkeeper or a petty trader, or to form a marriage

portion for the maid. It may be from the old sense of exile and depression that they try to parade before the world a fictitious poverty; or, more likely, as they are essentially Jews of the old school, and await hourly the coming of Christ, and expect to have to move any moment to Jerusalem, they desire to leave as little as possible behind. Whatever the reason may be, the fact remains that there is little in their houses worth having, whilst their women are laden with jewels and gold; and though they never own land, they farm everything in the province from year to year. So strictly do they adhere to the letter of the law, that they have a difficulty about the fourth commandment; but as the cow must be milked and the horse fed, they engage a Mahommedan to serve every Sabbath, and do not consider him a stranger within the gates. A Sabbath day's journey includes any wandering within the walls of the town, though the river must not be crossed. One superstition has crept into the religion of this peculiar people, for they attach almost Divine importance to fire. They themselves can offer no explanation of this, nor can they show any authority for it in their laws, but it is clearly derived from long subservience to and contact with the fire worshippers of old. All this only shows how a literal adhesion to the best and most stringent of sanitary laws fails when the spirit of the act is ignored.

I shall now turn to a far brighter picture, that of a Mahommedan household. There are certain ablutions ordered by the Koran which have been carried out in different forms, as the Mahommedans divided themselves into various sects; but it is curious that while the Jews have confined themselves to the letter of their sanitary laws, the Mahommedans, with less regard of detail, have acted up to the spirit of theirs, while both acknowledge Moses as a mighty prophet, and their highest sanitary authority. It is quite a pleasure to watch a Mahommedan family of the middle class, and I have special opportunities of knowing their inner life. Everything is so clean. Their couches are brushed and their carpets swept every morning. Ask for water, and it is brought filtered and sparkling in a clean glass. Their food is simple, and quite as wholesome and cleanly prepared as our own. When they retire to rest they do so in separate rooms, changing entirely, putting on night clothes and sleeping in sheets, which are quite as often changed and as frequently aired as our own. And now that bread is 40 lbs. for a rupee and meat 10 lbs. for that sum, and as unskilled labour is at ten rupees a month all the year

round, it will be seen that wholesome food is within the reach of everyone. The greater part of Mahommedan men drink moderately, and what is a recent improvement, not in secret, as before, but with companions. But very lately some of the highest ladies of the harem have taken to beer, from an idea that it will make them fat. The thoughts of Mahommedans are of cleanliness, and not a mere sense of obligation to obey some given law. Sometimes twice, but at least once a week, even the poorest of them are thoroughly scrubbed with soap and hot water in the bath. The large houses have baths attached to them. The public baths are open to all, and the system of payment is almost voluntary, for it costs the poor only a trifle. It is certainly an objection that diseased persons are not excluded from the bath, but as every one takes his own soap, towels, and scrubbing apparatus, and as the water is run in by a tap, and as soon as used run off again, the danger is not so great as if it were a plunge bath. The Mahommedan must not be judged by the Mahommedan of India, who is not a real Mahommedan, but half a Hindoo; and on his arrival here cannot sometimes for years shake off his early prejudices, and embrace his religion in its purity. The Mahommedan lady often does much household work; but as her house is well furnished and kept clean she requires servants. As the Mahommedan is entitled to four wives, all females are married, unless deformed, and there are not sufficient for the demand. Recourse has therefore to be had to African slaves; and the African female slave is certainly, considering her position in the human scale, now in no worse a condition than the English domestic servant. She has the privilege of making the family unhappy, should she desire it. She can always appear before the judge, and without trouble obtain her freedom for the same amount of cruelty that would separate an English woman from her husband. There is no law to force her to work, and, should she desert, there is now no law to make her return. Though aware it is not prohibited by the Koran, the intelligent Mahommedan knows that slavery is inhuman, and (I write only of this province) if he could obtain free labour he would prefer it. These descriptions of the Jew and Mahommedan will tend to show why other things, such as houses and drainage, or rather want of it, being the same, the Jews are attacked by certain diseases with greater severity than the Mahommedans.

This province having no history it is impossible to trace plague further back than the time when the Hon. East

India Company had a factory in Busreh, but there is no doubt that the disease has existed for ages, breaking out at certain times, for though very little of cuneiform literature has been read, Mr. George Smith has discovered the occurrence of three separate attacks of plague in Babylonia, one, 803 B.C., one, 765 B.C., and the third, 759 B.C., all these coming, as it will be observed, close together. The disease is written in the cuneiform as "mantana", which means, in the Arabic of to-day, the sweeping away of whole families, though it may be argued that this might occur in pernicious fever. In the fourth century of the Christian Era, plague broke out at Seleucia, the ruins of which lie on the Tigris some eighteen miles below Baghdad; the disease played havoc and went in the way of Syria. Men of Baghdad have told me that their fathers have told them that a terrible plague raged here about 1715, though the first authentic history of plague we have here is in 1772, when, by all accounts, it was exceedingly severe; again in 1800-2, and in 1831-4. I have already given the history of these plagues in the *Transactions of the Medical and Physical Society of Bombay*, 1871, so that I need not allude to them any further than to say that I believe there has been a good deal of exaggeration respecting them. They speak of deaths by the thousand, 3,000 and 7,000 a day in the Plague of 1773, and they say that in that of 1831, 60,000 people died in Baghdad alone. That the mortality was terrible I do not doubt, but such large numbers as these are not even within the grasp of Europeans. It is to be remembered that during the plague of 1831 in Baghdad, water-carriers went about supplying water to the houses nearly the whole of the time, and men could be got to drag the bodies of the dead into the river; now if we think that the disease existed in Baghdad only a month, and if we take the population as given at 150,000 (men, women, and children), it seems impossible that 60,000, or anything like it, out of the 150,000 could have been dragged into the river in that time; seeing that if 60,000 died, the same number must have been attacked and recovered, as the mortality was half the attacks. Be it remembered that this is still the land of the Arabian Nights; that even Europeans who have been some little time here cannot resist making a good story; and that fear would doubtless add to the supposed mortality.

Undoubted plague appeared in the marshes of the Hindeah in the spring of 1867. Namek Pasha appointed a commission to go and examine it, and also invited me to accompany it. I was president of the commission, and no one

on it had the slightest doubt but that the disease we saw was plague. It is unnecessary to mention it further than to say that it disappeared in the beginning of June. In 1874 plague showed itself in groups of villages at Dararah and Affiche, and was imported from there into Divanyeh, where 400 people, including the Kaimakan's son, out of a population of 2,500, died. I do not, however, state this as a fact which I saw with my own eyes, and I suspect there may be exaggeration in the number of deaths. From Affiche it was carried in the spring of that year to Um Nejeris, the villages of Dararah, Affiche, and Um Nejeris being of the fourth class. The disease then became dormant on account of the great heat setting in, but in the autumn of that year it reappeared slightly at Um Nejeris, becoming afterwards once more dormant till the beginning of January, when it was reported to exist on the river Hai. It was then carried to the district of Abdair, which lies by the canal or river, I do not know which to call it, leading the waters from the Affiche marsh to the Euphrates. Immediately after that it again appeared in another marsh of the fourth class called Fowar. It then again threatened Divanyeh by direct importation from Fowar, but only killed a few people on the outskirts and did not enter the town. From the river Hai the disease was also imported into Nassreah, but few people were attacked. It then went on to Sook-il-Shivok, were 200 people died out of a population of 2,000. At this time it again became dormant. In my description of the towns, I have omitted entirely mentioning Sook-il-Shivok, because I was unable to place it under any of the three divisions from my ignorance of its history. But this Sook-il-Shivok, which was the capital till Nassreah was built, of the Montafik country, and is a purely Arab town, is as filthy as any town I have seen.

In November 1875, the disease again broke out in the permanent marsh-village of Obara, which lies ten miles north north-east of Hillah, and out of eight families only one old woman remained. Not far off was the village of Albu Jassim, containing 400 inhabitants; and here in six to seven days eighty people died. From these villages it was traced into the town of Hillah, which lies on both sides of the Euphrates, and where it remained a whole month on the left bank before it went over to the right. On its appearance in Hillah, many families who could afford it flocked into Baghdad, and the people of Baghdad, after my visit to Hillah, anxiously watched the progress of the disease and dreaded its importation. The excitement rose to such a pitch that the Sheas spread a story that a horseman with a

spear met a caravan on its way from Hillah to Baghdad, and demanded an old woman who accompanied the caravan. The muleteers said she *was* certainly here, but had now disappeared; the horseman then rode through the caravan and speared a donkey, when the old woman appeared from inside the donkey and he killed her. The horseman was Ali, son-in-law of Mahommed, and the old hag represented Plague, so that hopes were raised that the attack might be averted from Baghdad for a season; but on the 13th of March 1876, it was officially announced by the quarantine that plague had appeared in Baghdad on the right bank of the Tigris, that is, on the side towards Hillah. The disease within the walls of Baghdad has confined itself chiefly to that portion on the right bank which contains about a quarter of the population; but on both sides within the city walls 4,585 cases have been registered by the sanitary commission, and 2,556 deaths. It is not to be supposed that this represents the actual number of cases and deaths, which were never registered. As I desire to confine myself to facts, I have no suggestion to make as to the percentage which ought to be added for unrecorded cases and deaths, but would remark that all the cases registered by the commission were examined by it. In the town of Hillah alone quite 4,000 people, at the lowest computation, must have died; this I have on the best authority that could be obtained, viz., that of a careful and intelligent English medical gentleman, who visited not only Hillah after the epidemic was over, but also Nejef and Kerbellah; and came to this conclusion from the average number of dead bodies that were taken from Hillah to Kerbellah and Nejef, as well as from what he himself ascertained in Hillah. Now if we consider that Hillah contained 15,000 or 16,000 inhabitants, this would give a quarter of the population, and on my expressing my surprise at so large a proportion being cut off, he told me I could form no idea of the desolation there. I will say nothing further of the cases and deaths till I have considered the symptoms of the disease.

Before doing this, however, I should like to say somewhat of the tables I have made out to illustrate them. As I said before, there were registered 4,585 cases, and 2,556 deaths, but a great many of these cases are simply registered as "attacked" and "dead"; while, in very many others, no indication is given of the duration of the disease, the regions where the glands were enlarged being only marked. I have, therefore, in making out the tables appended, taken from the whole register cases only 534 in number (*vide*

Table I), in which the duration of the disease is given; and as it has been impossible to work up all the 4,585 cases, I have taken, to illustrate the position of the glands most frequently attacked, that quarter of Baghdad on the right bank of the Tigris, called Fahama. This was said to contain 2,000 inhabitants, and is now declared by the inhabitants themselves to contain only 1,000. According to the register, the cases there were 471, and the deaths 359. In mentioning these numbers to the petty authorities, they laughed and said, "We gave only as few as we could venture to do." In that quarter I consider that the actual deaths must have been at least as many as the registered cases were, that is to say, 471. This Fahama was attacked with great severity, and its proportion of deaths would rather represent what is met with in an outbreak in one of the marsh villages, than what is the ordinary behaviour of plague after its importation into the larger towns; for plague, at its height in the marsh villages, has destroyed from 93 to 95 per cent. of those attacked. I desired also to extract from this register the proportion of attacks and deaths to the number of the household, but the data I have do not enable me to do this with accuracy, though perhaps it may be done next season. Sufficient it is to say, that when plague entered a house, I mean those houses of the second class in which the people live on the ground, it clung to it, and few escaped unless they deserted the place.

Symptoms.—In the beginning, this disease is often not to be distinguished from ague; though sometimes before fever sets in, the patient, instead of merely complaining of pains in the limbs, which are the premonitory symptoms of ague, becomes distracted, tosses about in evident fear of something he cannot describe, and if he is absent from his house runs home like a maniac, and throws himself on his bed. The fever which sets in lasts from twenty-four to thirty hours, his eyes become red and turbid, his tongue appears swollen and fissured, sometimes black, sometimes white, as if cotton were upon it; he has to be spoken to twice or three times before he can understand the question, he will then probably sit up, if he can, before answering, and his head will roll from side to side, and his face become flushed like that of a drunken man. Sometimes, no answer is to be got to a question, however often it may be repeated, or if an answer only a moan. Sordes are invariably about the teeth and gums. If the patient can speak, he demands water, for the thirst is intense, and he will probably complain of a pain, as if a knife were stabbing him in the epi-

gastric region, or as if it were full of burning charcoal. Both last year and this there has been comparatively little vomiting, and when it did appear it was generally bilious, though sometimes bloody: and in the case of one patient, I particularly noticed when she vomited blood, at the same time blood came from the rectum and vagina; it is unnecessary to say she sank rapidly. During the fever stage the breathing is hurried, the pulse very rapid, and the temperature of the skin about 100 degs., while the thermometer in the shade is 93 degs. With the fever, or oftener some hours after it has set in, the lymphatic glands enlarge; but I have known cases in which the patient, apparently in good health, has screamed out that he has been stabbed in the groin. There are cases in which the glands enlarge *before* the fever sets in, and they are quickly fatal. As will be seen by a table appended (*vide* Table II), the glands which chiefly enlarge are those of the groin, next those of the axilla, after that those of neck; and I have seen two or three cases of the gland in the bend of the elbow being swollen, but never one instance of enlargement of the popliteal glands. On occasions, the glands in more than one region enlarge; these glands, in enlarging, are either oval or round, one in a string being larger than the others, which gradually diminish as they recede from it, the largest gland being sometimes the size of a small orange. Till now, I had imagined that carbuncles were a favourable sign: I see, however, that in eleven cases registered in Fahama, in which carbuncles appeared, nine died. The urine is generally natural, abundant and pale-coloured. Generally, there is obstinate constipation; diarrhœa, when it occurs, is considered a favourable sign. Petechiæ are, almost without exception, to be considered as fatal signs; they appear, generally, only a few hours before death. Sometimes there is a return of the fever, which then becomes intermittent, but I do not consider that to be a return of the fever of plague, because every disease in those parts, whether fatal or otherwise, generally terminates in an attack of ague. I have mentioned that, taking the whole of the register, there were 4,585 cases, and 2,556 deaths. This gives a proportion of deaths to attacks of 55·75 per cent. of the cases: 2,165 are males, and 2,420 are females. Of the deaths, 1,134 are males, and 1,422 are females (the percentage of male deaths to attacks being 52·38, and that of female deaths being 58·74). I do not know how to explain why a greater number of females should have been attacked, for though at first sight it might be supposed that as this is a Mahom-

medan country the explanation is easy, still as I have previously shown, few of the poor have more wives than one, and this is a disease which has essentially attacked the poor, not one per cent. of those attacked having lived in houses of the better class. In Table II, appended, I have endeavoured to show the age at which a person is most liable to be attacked; but the numbers are insufficient for more than mere general conclusions. The fœtus has been in some cases attacked, and in one case of premature birth which I saw there were twins, one child being dead, with the glands in the left side of the neck swollen, while the other child is now alive and doing well. It will be seen by Table I, appended, that of 534 deaths 311 died within the first three days, and that then the risk of death gradually diminished as the days went on, so that the natives themselves were pretty correct when they said that a patient may consider himself safe after the seventh day. I think that the proportion of about 60 per cent. in the first three days is much under the mark, for I suspect that many, if not most, of the cases entered in the register as simply "attacked" and "dead" are those of people who died in that time. It has been said that the strong and healthy are more liable to be attacked than the sick and the weakly; but that is a point I am unable to settle till I know what proportion the sick and the weakly bear to the whole population.

Having described the symptoms of the disease, I ought now to say a word on the treatment. It is almost unnecessary to say that no cure has as yet been discovered, but as cases in which diarrhœa early sets in are looked upon hopefully, a purgative where constipation exists is indicated; and when the actual fever leaves, as depression and exhaustion quickly follow, I think that stimulants ought to be given. Not under any doubt as to this being plague, but under the hope that it might be of some use, quinine in large doses, sometimes combined with carbolic acid, was given freely by the Local Sanitary Commission, but every member acknowledged it was utterly useless, and my impression is that it was rather injurious than otherwise. The Turkish troops were largely dosed with quinine, and they had to take it on the first appearance of febrile symptoms; but of those attacked by plague 50 per cent. died. As there is no immediate treatment, it becomes a serious question what can be done to check the progress of the disease: and in considering that point it becomes absolutely necessary also to consider what assistance can be got from the Local Government and the people themselves, and to find out

how far measures of undoubted good can be forced upon the people, and how far the Government is able and willing to support those measures. In this case the Local Government has simply ignored the existence of plague, and caught at any straw to escape confessing it, such as that this is a swamp fever easily cured by quinine, and that it cannot be true plague, because it does not at once play havoc. But if the people would only think that in this one quarter of the town called Fahama about a quarter of the people died, their own account indeed being (not a mere story but from calculation) a half: and if they would only picture to themselves the disease attacking the whole of Baghdad as it did Fahama, then there would have been no question as to its true nature. The Sanitary Commission at Constantinople, though in hourly communication by telegraph with Baghdad, delayed till the epidemic was well advanced before issuing orders. Then came an order for whitewashing and fumigation of houses, for cutting off communication between towns and villages, and the breaking of bridges; and they had previously imposed local quarantines, which I hope presently to show are worse than useless. The Local Government also then exerted itself, but the people threatened to resist, for simple minded though they are, they saw how useless, inconvenient, and actually injurious those measures would be. I have tried to show in the photograph of the plague-house what whitewashing here means: it consists in dipping the hand or a pocket handkerchief in whitewash, and passing it once or twice along a bit of the wall. Fumigation consisted in burning a little sulphur in the courtyard, and often in merely giving the sulphur to the household to be burned. The result of the professed cutting off of communication between towns and villages raised the price of grain: but how far it was in the least degree effectual may be judged when I describe the local quarantines. The result of breaking the bridge of boats joining the two divisions of Baghdad was simply to give great trade to their round basket boats called "Kufas" after dark. The attempt to shut up people in houses in which anyone had been attacked was simply absurd, because there is about as good a road along the flat roofs from house to house as there is in the filthy passages dignified by the name of streets. The Local Commission has acted during this epidemic with judgment. It kept a register, imperfect certainly, but excellent so far as it went, of all the cases that came under its notice within the walls of Baghdad. As it was composed chiefly of men who knew what could and what could not be done, of men who desired to do their work

honestly and give no unnecessary annoyance to the inhabitants, that is to do no evil, it saw the absurdity of local quarantine, but could not help it, and knew that "cordon sanitaire" had become a bye-word in the place. It knew that to attempt disinfection by whitewashing and fumigation would, if it did not create a rising, at any rate prevent its getting any returns, and it saw the absurdity of shutting up families in houses with flat roofs. This commission watched the disease and had the courage to say that it could do no more while the epidemic prevailed. When the epidemic ceased it immediately proposed that the villages of the fourth class, previously described, should be removed some few hundred yards from their present site, that is, out of the filth; it also proposed a thing of great importance, viz., that so soon as one was attacked in a house with plague, the healthy members of this family should be removed, groups of reed huts being placed round the town to receive them; and it further proposed that a small committee of active men should be formed in the various towns to carry out arrangements of cleanliness during this season of repose. The Sanitary Commission at Constantinople telegraphed to know what the cost would be of shifting the marsh villages, and I fear that question of money will be at the bottom of much evil; for I suspect nothing will be done this year, although those villages of the fourth class could be moved with little more trouble than it takes to shift an encampment of tents.

I consider myself a great authority on the local quarantines established in this province, because it has been my misfortune to be caught in them no less than three times. Once it was for cholera, with some 5,000 Persians, Afghans, and Oriental Russians, herded together without the slightest care near the town of Khanakin on the frontier between Persia and Turkey. This, my first experience of quarantine, certainly did open my eyes to the evil it could do, even if it also did any good; but it did *no* good, as it happened, for while these men were collected cursing the Turks, it was well known in camp that cholera had appeared in the villages between that and Baghdad, and that predatory hordes passed and repassed the Turko-Persian frontier as well. It was in fact soon seen (for these men can reason as well as ourselves) that that quarantine at Khanakin was simply established to get into the coffers of the Sanitary Commission at Constantinople the £15,000 which was then yearly cleared. I saw the end was near, though I did not think so quickly; for it was only a year after-

wards that the Persian Government prohibited their pilgrims from entering this province, a move which has almost ruined Baghdad, and deprived the Commission of its income. In fact the Constantinople Commission killed the goose that laid the golden eggs, for it supposed that when cholera was reported at Resht on the shores of the Caspian, it might with impunity impose a quarantine here. The second time I was caught in quarantine was close to Baghdad, for plague, in June last year. One might think that if any internal quarantine could be strictly enforced, it would be on board a river-steamer, but when I arrived in one vessel, another was there before me, and many of its passengers were already enjoying themselves in Baghdad. There were many Jewesses on board my steamer, returning from a pilgrimage to the tomb of Ezra. A Jew complained to me that while during the first evening or two poor Jewesses coming out from Baghdad in exchange for their wealthier co-religionists on board required only five shillings for their trouble, now they wanted a sovereign, or to be more correct a Turkish lira. All Jewesses, rich and poor, are wrapt in sheets dyed in indigo, and it is impossible to recognise them. I saw a servant of mine bearing on his shoulders in broad daylight a mountain of dirty linen, which I had been wearing while in search for plague on the Euphrates. No one cared, and on my remonstrating, my head-servant told me that he had arranged with the guardians. I ordered the clothes back to the steamer, but when I reached Baghdad, I found them washed and ironed ready for use. My third experience of quarantine was this year near Kurnah, where it was established on the plea of protecting Busreh. The quarantine station was not placed below the junction of the Tigris and Euphrates as one might imagine, and where high ground could easily be got, but on the Tigris, alongside of a swamp with rice fields beyond. About twenty passengers had been foolish enough to enter the jaws of quarantine in a Turkish steamer; but while wool was flowing freely into Busreh there was not an ounce in quarantine, and travellers generally simply diverged to right or left and entered Busreh with impunity. It may be well said that if internal quarantine is out of the question, it would be easy enough to establish quarantine at Busreh for vessels from sea-ward. But, be it remembered, that Busreh is seventy miles from the mouth of the combined Tigris and Euphrates called the Shat-il-Arab, and that the left bank belongs to Persia, while the right only is Turkish. Steamers and square-rigged vessels it is true are boarded at the mouth,

and guardians put on board; but while these clean and well-ventilated vessels are carefully watched by the quarantine authorities, most of the native craft are below their notice, and there is no obstacle at all thrown in the way of people crossing the river from Persia to Turkey. This then is, as fairly as I can give it, my experience of quarantine here; and I cannot understand, unless indeed this province is looked upon as a milch cow to feed the demands of Constantinople, why intelligent men should continue to impose quarantine here. It is worthy of particular remark that every time plague has reappeared for the season it has done so in a permanent marsh village, and has been imported into the towns; and if this is to continue, and if plague is to retain the same rate of progression, there is no fear either for Europe or India; because, west of Baghdad the marshes cease, and eastward they go no further than the top of the Persian Gulf. But this is a point on which I fear it would be unwise to lay too much stress. We must rather look upon plague as a growing boy, and if nothing providentially happen (for people here are not likely to help themselves) he will grow into a man; and he has already shown what he can do in small places as a boy, so that I confess I feel anxious as to next season (1876). It would at first sight seem that as plague kills only a certain number who may be liable to it, and as it appears at intervals of about thirty years, when a new generation has arisen, therefore it simply lies in wait for a fresh batch of victims; but if this were the case, as it attacks the infant and the fœtus in the womb, it would become an endemic children's disease. The question of contagion must be left for the present; whole families have in very many instances been swept away, but the disease has not as yet proved itself contagious in the sense former epidemics are said to have been. We must also hold, I think, to the belief that skins, hair, and wool are capable of bearing contagion, until we can prove distinctly to the contrary; because it would be unwise and inhuman to risk the lives of human beings and other countries for the sake of giving a little more money to a handful of merchants.

TABLE I.—Showing the Duration of 534 Cases of Plague at Different Ages.

Age in Years.	Sex.	Time in Days of Death after attack.													Total.
		1.	2.	3.	4.	5.	6.	7.	8.	10.	12.	16.	20.	Over.	
1 and under	Males	2	1	1	—	—	1	—	—	—	—	—	—	—	5
	Females	4	1	2	1	—	—	—	—	—	—	—	—	—	8
1-12	Males	17	12	15	11	11	3	2	—	—	—	—	2	—	73
	Females	23	22	21	15	7	5	—	1	4	—	—	5	—	103
12-30	Males	24	15	15	11	12	7	1	5	3	4	—	—	—	97
	Females	24	9	18	14	9	5	6	4	4	1	—	1	—	95
30-40	Males	5	5	7	4	4	1	1	—	—	1	—	1	—	29
	Females	14	5	9	7	7	2	—	2	—	1	1	2	—	50
40-60	Males	2	5	6	4	1	—	—	1	2	1	—	—	—	22
	Females	9	5	7	5	7	2	1	1	1	1	—	—	—	39
60 and over	Males	—	—	3	2	1	—	1	—	—	—	—	—	—	7
	Females	2	—	1	2	1	—	—	—	—	—	—	—	—	6
Total	Males	50	38	47	32	29	12	5	6	5	6	—	3	—	233
	Females	76	42	58	44	31	14	7	8	9	3	1	8	—	301
Grand Total		126	80	105	76	60	26	12	14	14	9	1	11	—	534

TABLE II.—Showing, of 402 Cases of Plague, the Numbers attacked at Different Ages, and the Frequency of Enlarged Glands (Buboes) in Various Regions and of Carbuncles.

Age in Years.	Sex.	Frequency of enlarged Glands in various Regions.								Carbuncles	Total.
		Neck.	Axilla.	Elbow.	Groin.	Crural.	Other Regions.	More Regions than one.	Glands enlarged; position not mentioned.		
1 and under	Males	1	—	—	—	—	—	2	—	—	3
	Females	—	4	—	—	—	—	—	2	—	6
1-12	Males	9	32	—	42	2	1	2	—	3	91
	Females	5	27	—	15	—	—	1	2	3	53
12-30	Males	5	32	—	33	4	1	4	2	2	83
	Females	4	25	—	34	1	—	—	—	1	65
30-40	Males	—	12	—	24	—	—	1	—	—	37
	Females	—	5	—	? 15	—	—	—	—	2	? 22
40-60	Males	1	7	—	9	1	—	3	—	—	21
	Females	—	8	—	5	1	—	—	—	—	14
60 and over	Males	—	4	—	2	—	—	—	—	—	6
	Females	—	—	—	1	—	—	—	—	—	1
Total	Males	16	87	—	110	7	2	12	2	5	241
	Females	9	69	—	70	2	—	1	4	6	161
Grand Total		25	156	—	180	9	2	13	6	11	402
Recoveries		6	47	—	52	—	—	5	—	2	112
Deaths		19	109	—	128	9	2	8	6	9	290

ON THE STUDY OF MEDICAL METEOROLOGY, ESPECIALLY IN ITS RELATION TO EPIDEMIC MALADIES.

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(*Read: February 8th*, 1876.)

I HAVE been honoured by your President with a request that I would this evening address you upon the study of meteorology in its relations to that branch of etiological research which gives its designation to this Society. The compliment is one which I am conscious of not deserving in any way, and, while responding to it, I wish in all sincerity to start by disabusing your minds of any expectation you may have fostered of learning from me to-night anything novel upon this very interesting and very important subject. I am painfully conscious of my unfitness to speak upon it with authority.

We must begin, where all true reformation begins, with an ample expression of our ignorance and shortcomings. We live in an age of precision, precision of observation, logical and strict precision in reference to all scientific questions. It is through precision that all the grand advances in our scientific knowledge, and all the great practical reforms resulting from the application of this knowledge to the exigencies of our daily life, have during the last half century been attained. And what this advance has been all can judge who are old enough to carry back their memories for one half of that period of time. Can it be said that our acquaintance with the bearings of meteorology upon public health, and especially upon the origin, spread, and decline of epidemic maladies has advanced commensurately with our acquaintance with other branches of etiological science? Let me take an instance, and I will select for illustration what has appeared to me to be the purest example of an epidemic malady, a malady which, while spreading from time to time at most uncertain and indefinite intervals over a great part of the world, does not appear to owe its spread in

a material degree to the operation of what we term contagion. You will probably anticipate me when I say I refer to the disease which we here call "influenza". Let us consult all available records of the numerous epidemics of this disease; let us put together, and with all the care we can, estimate the value of the statements given us of the meteorological conditions prior to and during the outbreak of the epidemics and at their decline, and what does it amount to? A tangled skein of apparent contradictions. And yet, if there be a law governing epidemicity, if outbreaks of influenza be not exceptional instances of non-coherence among mundane phenomena, there can be no such contradiction in point of fact. It is unquestionable that remarkable meteorological conditions really have preceded and accompanied these outbreaks, and it would be folly to assume that they had no etiological relation to them whatever. It remains for modern enquirers, with greater facilities for their enquiries than their forefathers possessed, to determine what the relation is. And let us not forget that, in doing this for influenza, it is not unlikely that we shall be taking a most important step in unravelling the complication of questions which still surround the epidemicity of other diseases, such as cholera, the specific fevers, and the epizootics.

I have mentioned influenza first because it appears to me to be the field of etiological research which in its meteorological aspects promises to yield the most abundant harvest of valuable results; but it is an epidemic which happily occurs with comparative infrequency. There are other much more common maladies, the epidemic prevalence of which may be more readily studied and perhaps with the prospect of earlier fruit. I allude to those common diseases which, at short cyclical periods, spread over this land of ours; but the study of which is, so far as meteorological etiology is concerned, somewhat complicated by the fact of the contagiousness of some of them. There are the acute pulmonary affections, which cut off so many of our young and old in the winter months, the diarrhœal affections which destroy our infant population every summer, whooping cough, small-pox, measles, scarlet fever, what we call the "continued fevers", and such rarer epidemic diseases as diphtheria and cerebro-spinal meningitis. And I mention these diseases because they are at our very thresholds, and every practitioner in the kingdom who pleases to do so, can lend a helping hand in their etiological investigation. What do we know about the meteorological conditions which influence the epidemical spread and epidemical decline of these diseases? Surely

they are frequent enough for us, if we had used all the means we might individually and as a nation have used, to have by this time acquired a very respectable acquaintance with the facts about them. I will not say we know nothing, for, thanks to the Registrar-General and his indefatigable and zealous coadjutor Dr. Farr, we do know something, and through their exertions we have in our hands the means of knowing a great deal more. But we want a Humboldt. And even if we had some one fitted by industry and previous acquirements to grasp with a master's hand the whole maze of facts which have thus been put within reach, and possessing withal the unvarying patience necessary to analyse these facts and to put them in order, we should merely arrive at something which would not afford us all the desired satisfaction. For the Registrar-General deals with deaths, and death from disease is only one phenomenon in its course, and an uncertain, and where we compare one epidemic of a disease with another, and the same epidemic at different periods of its course, a most variable one. I would not even for a moment insinuate a shadow of a doubt as to the immense value of the medico-statistical records issued by the great national office presided over by the gentlemen I have named; but they will probably be ready to admit that for the satisfactory solution of the great questions of medical meteorology now awaiting solution, something more is wanted than records of mortality. What we sorely need, is a record of sickness standing upon such broad basis as are our records of mortality. But I am anticipating—pardon me. I ask, what do we know with precision about the common diseases I have enumerated? Am I wrong in saying that while the Registrar-General has familiarised the public mind with the fact that cold weather enormously increases the mortality referred to pulmonary diseases, notably to bronchitis and pneumonia, and that heats of summer are associated with greatly increased infantile mortality from diarrhœal affections, we are yet in ignorance why, in years or corresponding months of similar winter cold and similar summer heat, the mortality from these diseases respectively is sometimes so different as it is; and as respects diarrhœal affections, we are yet in doubt whether it is temperature, or some other meteorological condition combined with it, which is operative in producing the observed result: and whether the result, so far as we know it, is the direct or indirect effect of the conditions referred to. And again, as respects the specific febrile diseases, we are at a loss to understand why, with the specific contagium probably never altogether absent from our midst,

it should operate epidemically at one season of the year rather than at another, and what are the precise meteorological conditions which promote or avert the epidemic spread and promote or retard its decline. Let us ask ourselves if we know why small-pox for instance spread epidemically as it did so recently, and why it did not spread epidemically in the interval between the last and the previous epidemic, notwithstanding the presence of an abundance of foci of contagion. Is it not for us to enquire whether atmospheric conditions had anything to do with the difference? Let us ask ourselves what we know of the meteorological associations of epidemics of whooping-cough, measles, and scarlet fever? Surely these are common enough. It is true that some of them appear cyclically—measles notably so—but what is there that governs these cycles, and what meteorological conditions determine the exact period of the epidemic outbreaks, their duration and cessation? These and similar questions are as yet unanswered. And we have further to learn what these conditions are, probably in part meteorological, which determine the fatality of epidemics—in other words, the severity with which they fall on the individuals attacked. Here is another most important branch of enquiry; for it is certain that there are mild epidemics at one time, severe and very fatal epidemics at another time, of all our common epidemic maladies. Do we know? I for one should be very glad to learn. So far as my own trifling observations go, they have given me a peep behind one corner of a curtain the full raising of which would, I am satisfied, disclose to view some facts of immediate practical value if not to the treatment yet to the prognosis of disease.

So much for our ignorance. Now for our shortcomings to which our ignorance is due. There are three requisites for a comprehensive and successful study of medical meteorology, and these are accurate observations at a sufficient number of meteorological stations, reliable records of sickness and its fatality, and a sufficient staff of workers to analyse and compare facts and work out results. The first we have in great measure supplied to us by our great meteorological societies; the materials for the second requisite exist in an imperfect condition in the records kept by Poor-Law Medical Officers and at public medical institutions, but they are not kept universally as they ought to be, nor have they been utilised as the more perfect of them might have been; the third requisite remains to be found. What we should, in my opinion, aim at first of all is as perfect a system of registration of sickness as is practicable

under existing circumstances of professional education. Towards the attainment of this, the Medical Officers of Health throughout the kingdom might if they pleased contribute most valuable aid. An acquaintance with the amount and character of prevailing sickness is essential to them for the due and efficient exercise of their office. One reason, I believe, why records of public sickness are not properly kept now is that the persons who ought to keep them have not seen the use of keeping them. If the information were sought regularly and were seen to be applied to a useful purpose, I cannot think that it would not be forthcoming. I have myself made the effort in the small district of London, which was formerly under my sanitary supervision; I met with difficulties, as a matter of course, but I surmounted them one after the other, and for several years kept on foot a tolerably satisfactory register of public sickness. I can, therefore, speak with some experience upon this subject. There is no insurmountable difficulty in collecting facts as to sickness, sufficiently precise for practical uses, provided the collector sets about the business discreetly and is sufficiently patient, persistent, and forbearing. Very much more might have been done by local observers, especially by official observers, than has been done in this direction. But, when the facts have been collected (I speak again from experience), the labour of analysis is such as no man engaged in professional practice can hope to be able to supply. It was here that I myself broke down. It is this, I believe, which, more than anything else, has served to discourage the pursuit of medical meteorology, in itself one of the most enticing of studies. What then, is to be done? For amendment ought to follow upon confession of our errors. I am addressing the Epidemiological Society, a Society which, as your President pointed out to you (and I think triumphantly) in his opening address, has already been the pioneer of great practical reforms. A grand opportunity lies before you, an inauguration of a new activity, opening out the prospect of advancing etiological science into an almost virgin field. To whom could the appeal to march on be addressed more appropriately than to you? You have already shown to the country and to the world what the unsupported efforts of a band of determined men can effect in overcoming obstacles and demonstrating the practicability of investigation in one direction. You have been successful beyond your own expectation, for the nation has undertaken that which you showed to be feasible, but could not yourselves have accomplished. Perform the same excellent *rôle* for medical me-

teorology—if you fail yourselves to effect all you aim at, you may still see before you the prospect of inducing our National Governors to take up your work and to prosecute it to a satisfactory conclusion.

I have hitherto confined my remarks to the epidemic diseases common to this country, and I have done so purposely; for it is abundantly clear that this work must be commenced at home, where the facilities of enquiry are most readily attainable, and where home sympathies can be enlisted in its behalf. But you cannot stop there, nor can you even be strictly limited, however you may desire it, to home observations. And here again it appears to me that, with your already organised foreign correspondence, you are especially fitted for the task I suggest you should undertake. For these common home maladies, when epidemic, are not only epidemic in England. They have a march, passing through other countries to arrive here, exerting concurrently in foreign lands and invading other nations after they have done their fatal work among ourselves. And to draw inferences as to our own epidemics, and their relation to local meteorological conditions alone, might, and probably would, lead to serious mistakes. It would be for you to endeavour to organise similar observations abroad to those you may organise at home. It may not be in your power, without such a paid staff as circumstances would probably forbid to such a small Society as this, to do much more than collect facts; but, in the absence of any systematic collection of facts at present, this would be doing a great deal. The time will surely some day come, when the facts you may have gathered will find their expositor. You will, at any rate, have shown the way to overcome the first obstacle to a true medical meteorology.

It is not for me, on this occasion, to suggest further than I have done in what way it would be most advantageous to set about the collection of sickness statistics; it is enough to say that all at present practicable is the collection of statistics of what may be termed public sickness, that which seeks medical relief at the hands of poor-law medical men and the various public institutions established for giving medical relief to the needy. It is a subject which has occupied the thoughts of many men more fitted for consultation upon it than myself. All I desire to say now is that the registration should include all cases, both surgical and medical, and not be limited to the classes of disease liable to prevail epidemically. And for this reason, namely, that it may be that certain atmospheric conditions so affect the

human constitution as to lessen its power of resistance to *all* causes of disease. I would not exclude surgical cases from enumeration, first, because it may be difficult always to do so, and secondly, because it is probable that the error thus introduced would be one of uniform magnitude, whilst it is probable that surgical diseases are influenced equally with diseases usually considered medical by conditions of weather.

The final object of the study, apart from the practical application of the truths to be evolved, is, not merely to ascertain the fact that such and such conditions of atmosphere are favourable, or the reverse, to epidemic extensions, although this is the first thing to bring out, but how it is and why it is that such is the case. We may say that it is certain even now that weather and season do influence in some way the spread of epidemics. Putting aside respiratory diseases and diarrhœal affections, about which nobody can for a moment dispute, I have shewn in an analysis of some thousands of cases recorded in my old health district at Islington, that during twelve years while measles, for instance, occurred with regularity as an epidemic once in two years, the greater proportion of cases happened in the months of May, June and July, and that there was exhibited another like tendency to epidemic spread in the months of November, December and January. This has no reference to the mortality. I am speaking of cases. It appeared also that there was a mean temperature, namely between 43 deg. and 49 deg., which favoured the epidemic spread of the disease both in the spring and autumn, and further, that a temperature above 60 deg. and below 42 deg. tended to arrest the epidemic spread of the disease. Again, as respects scarlatina, the fact came out most distinctly, that this is *par excellence* a disease becoming epidemic towards the beginning of the summer quarter. The smallest number of cases was observed in the month of March, there was a slow and steady increase to June, the month of July showed a rather sudden increase in the number of cases, and the largest number occurred in the months of September, October and November. Again, I am speaking of cases not of deaths, for the fatality of the disease, at its minimum in the summer, arrives at its maximum in the cold and damp weather of autumn and winter. Mortality tables are specially unreliable as indicating prevalence in respect of this disease. With respect to temperature as influencing prevalence, it appeared to me that a mean temperature between 56 deg. and 60 deg. was that most favourable to an epidemic outbreak, that a higher tem-

perature was not unfavourable to it, but that a fall of mean temperature below 53 deg. tended to epidemic arrest. Small-pox, in my Islington experience, presented the smallest number of cases in September and October, and the largest number in March, April and May, the graduation of cases in intermediate months being tolerably uniform. This is clearly a disease the extension of which is promoted by cold weather and checked by hot weather. Very different this to popular opinion and to opinions I have heard openly expressed by some medical men. It is not true, as some have supposed, that all zymotic diseases are promoted by hot weather, and checked by cold weather. It appeared to me that a mean temperature of about 52 deg. was an important temperature as respects small-pox; a temperature below this promoting its spread, and a higher temperature checking it.

Now I beg to interpose a caution. I am not putting forward these few observations as settling the question raised as regards these diseases. I am quite aware that they were not all made in perhaps the best possible way, and that even if they were absolutely true for Islington, they might not be similarly true for the whole of London, for the whole of England, or for other countries. I merely adduce them to show that seasons and atmospheric conditions, do in a definite manner promote or check the epidemic extension of these common maladies, and that these conditions are, with the exercise of proper care and due patience, capable of being ascertained. The next question will be how they operate.

In considering this, when we are dealing with diseases of a contagious character, we have to keep in mind that conditions of weather and seasons may operate by their influence upon the functions of the body which is open to receive the contagion, by leading people out of doors where they mix with others, or by keeping them within doors and huddled together, or finally by their influence on the contagion or morbid product which is the agent of contagion. It will have to be determined in which, or whether in all, of these ways, atmospheric conditions operate in promoting or checking each form of contagious epidemic disease. And probably all these modes of operation may be ascertainable in respect of diseases not regarded as contagious, such as influenza. For although a disease may not be contagious, in the sense that the morbid material is not reproduced in the subject attacked, and so is not communicable from them to the healthy, it by no means follows that there is no

materies morbi at all in question. It may be that there is one which has some nidus in which it propagates itself other than the human body, such as the bodies of the lower animals or something not living at all, such as water, the soil, or the air itself. See then where you are being led, and how this field of investigation opens out before you.

And there is a yet further point to be ascertained at which I have hinted already, namely what influence is exerted by meteorological conditions in preparing the way for epidemic extensions. What is the reason of the cyclical recurrence of epidemics of which measles is so curious an instance? Is there anything similarly cyclical in the meteorology of the places where such cyclical recurrences are observed? For surely there must be some ascertainable reason for so remarkable a phenomenon. There must be some reason why contagious diseases fall into abeyance as epidemics for a period more or less decided in duration, but differing in different diseases. And what is it which determines the mildness or fatality of epidemics when they do occur? All these questions have to be considered from a meteorological point of view. Possibly their solution may be discovered when they are regarded on this side.

Above all things let us collect facts, and keep *a priori* reasoning within its proper sphere in these matters. But at the same time let us not be blinded, by the special study in which we are engaged, to the fact that there may be other influences, not meteorological, which govern the period of origin and decline of epidemics. I will merely mention one, "acclimatisation". Are there not facts on record leading to the belief that the human body may become, in time, so accommodated to the presence of morbid influences, as to become insusceptible to their operation? May not this, to some unknown extent, explain the fact of the cessation of epidemics after lasting for a time, notwithstanding the multiplication of contagious foci, and not merely, as is commonly said, the exhaustion of subjects who are susceptible because they have not previously suffered from an attack of the disease? Let us recollect the epidemic law of Dr. Farr, so strikingly illustrated by the cattle plague of 1865. Let us not forget also to enquire to what extent it is true that the intensity of operation of a contagious principle lessens by its repeated transmission through the bodies of men and animals.

I have now performed the task I had set myself; I feel that I might lessen the force of what I have written were I to enter into details. My paper is intended to be suggestive and nothing more.

ON YELLOW FEVER.

By Dr. STERNBERG,

Surgeon United States Army.

(*Read: April 12th*, 1876.)

[Abstract.]

The subject to which I am to invite your attention to-night is one in which I am sure you will be interested as physicians, and as epidemiologists, although as practitioners of medicine you are not, judging from past experience, likely to encounter in England the disease of which I am to speak. Your not having suffered from epidemics of yellow fever in England is to be attributed, chiefly, I presume, to the fact that your summer temperature is not maintained for any length of time at the point which seems essential to the development of an epidemic (about 72 deg. Fahr.) That the germs of the disease may be transported across the ocean, retaining all their potency for mischief, we have ample evidence in the epidemics which have occurred in the seaport towns of Spain. And I am informed that in two instances the disease has obtained a slight lodgment on your own shores. Once at Swansea in Wales, and once at Newhaven.

In the United States certain of our seaport towns upon the Atlantic and Gulf coasts have suffered repeatedly from epidemics of yellow fever since the earliest settlement of the country. The disease has prevailed, though rarely, as far north as Boston; but its ravages have been chiefly confined to cities in more southern latitudes, and especially to Charleston, Savannah, Penzacrea, Mobile, New Orleans, and Galveston. Following the course of the Mississippi river the disease has several times visited the cities of Memphis and Vicksburg, and has even penetrated as far into the interior as St. Louis. So lately as the year 1873 a very severe epidemic devastated the cities of Memphis and Shreveport, the former on the Mississippi and the latter on the Red river.

I shall devote the time at my disposal to a narration of the facts pertaining to several local epidemics which have occurred during the past five years under my own

observation, and to a discussion of these facts in their bearings upon the questions in which you as epidemiologists are most deeply interested—viz., on the origin, causes, and best modes of preventing epidemics of yellow fever. But first permit me to make a few general remarks upon the nature of the disease under consideration. Yellow fever is a distinct specific disease with well marked characteristics of its own. In a Darwinian sense it may perhaps be looked upon as forming a connecting link between the periodic and continued fevers. Like the former it is a disease of certain localities and seasons, and it is in those latitudes where periodic fevers exhibit the greatest malignancy that yellow fever has its permanent home. But unlike these fevers it is a disease of the cities and not of the country. In certain cases of paludal fever denominated in our Southern States pernicious remittent, the paroxysm of fever bears so close a resemblance to that of yellow fever that one disease is frequently mistaken for the other.

There are still physicians who have had considerable experience and who assert that yellow fever is a disease of local orgin not differing in its essential characteristics from the ordinary paludal fevers of the country, and that all attempts to exclude it by quarantine restrictions are unnecessary and futile. While there are striking points of resemblance between the paroxysm of fever in pernicious and congestive forms of paludal fevers and in yellow fever, yet there is this essential difference — that in the latter there is but a single paroxysm, which may be of two, four, or eight days' duration (*Vide* article by the writer in the *American Journal of the Medical Sciences*, number for July 1875), and that in the vast majority of cases one attack protects the individual from a subsequent one during the remainder of his life. It is this peculiarity of the disease which I especially referred to in remarking that it has some characteristics in common with the exanthemata. Other points of resemblance may be found in the tolerably definite period of incubation, which I have observed in many cases to be about five days. It may be, however, considerably less, and there are apparently well authenticated cases in which it has been much longer. Also in the clinical history of the disease, especially its mode of invasion, the tendency to visceral congestions, the presence of albumen in the urine, etc.

I must not neglect to mention the therapeutical fact that quinine, which is so potent a remedy in the periodic fevers, is now generally acknowledged to have no power to arrest the progress of an attack of yellow fever.

The yellow fever poison differs essentially from that of diseases commonly spoken of as contagious (using the word in its popular sense), in that it is not a direct product of the bodies of those suffering from the disease, at least, not in sufficient quantity or in proper form to endanger the health of those who are in immediate attendance upon the sick.

Epidemics of yellow fever do not arise by the transmission of the poison from person to person, either directly or through the medium of the air; nor, so far as we know, is the disease indirectly communicated through the medium of fluids imbibed, as has been so clearly demonstrated to be frequently the case in typhoid fever and cholera. How then, you will ask, is the disease propagated? I can find but one answer, viz., that the poison is capable of self-multiplication, independently of the human body; that it was indigenous in certain parts of America prior to the discovery of the country by Europeans; that these places are beyond the limits of the United States, where it may consequently be spoken of as an epidemic; that it is frequently transported to our shores in vessels sailing from ports in the West Indies, Central and South America, where it has a permanent home; that being so transported and finding, temporarily, conditions favourable to its increase, a minute quantity may multiply indefinitely and give rise to an extensive epidemic if it happens that a large number of persons come within the sphere of its influence; that unfavourable local conditions, especially as to temperature, affect either the development or potency of the poison, thus retarding the progress of an epidemic, and that a freezing temperature entirely destroys the poison, so that without a fresh importation the disease will not reappear.

I would not have you to understand that I consider man irresponsible for the dissemination of yellow fever, except in so far as he gives it free transportation in his ships from one port to another. On the other hand, I am convinced that he does play a very important part in promoting the progress of epidemics, not as a producer of the poison, but as a carrier of it from infected localities to others previously uninfected, thus establishing new centres where the poison may multiply by virtue of its own inherent properties in connection with surrounding local conditions favourable to it. The man who thus acts as a carrier of the yellow fever germs may or may not be himself a victim to the disease.

That filth, and heat, and moisture, and deficient atmospheric ozone, separately or combined, are capable of originating an epidemic of yellow fever in the United States, we

have no proof. On the contrary, we have the strongest evidence that they have no such power. When, however, yellow fever germs have been introduced, these agencies have, without doubt, a powerful influence upon the rapidity and extent of their development.

I shall now confine myself to facts which have come under my own observation:—During the late Civil War in the United States, I was for two years stationed in the city of New Orleans, and while there performed the duty of Secretary to the Board of Health under the military government of the city. At this time a large number of unacclimatized troops from the north were stationed in and about the city, and our hospitals were filled with soldiers who were suffering from wounds and from the diseases of the climate. None, however, were attacked with yellow fever, and I believe this to have been due to the restricted commerce of the city during the war, and to our efficient administration of the quarantine laws.

In the summer of 1871, while on duty at Governor's Island, New York Harbour, as the Junior Medical Officer at the station, a disease made its appearance, the diagnosis of which, for a time, baffled the combined skill of the two Medical Officers of the garrison, and of the Physicians of the Board of Health for the cities of New York and Brooklyn.

That it was a malignant epidemic was evident from the rapidity with which the sick list increased, and from the fact that several of the patients died after a very brief illness. At last we all arrived at the same diagnosis, at about the same time, viz., that we had to deal with yellow fever. The first cases were really as well marked as subsequent ones, and the only excuse for such a tardy diagnosis is to be found in the fact that none of us had previously seen yellow fever, and all were entirely unprepared for an outbreak of this disease on an island in New York Harbour, at a time when, so far as was known, no cases of the disease had occurred elsewhere in the harbour, nor was it prevailing anywhere on our coasts.

How yellow fever reached Governor's Island on this occasion, has never been determined with any degree of certainty, and the epidemic may to some appear to support the views of those who believe in the local origin of the disease as a result of certain atmospheric or telluric influences. There are reasons why such a view appears to me entirely untenable. In the first place, I cannot conceive any such influences to be confined to a small portion of this

island, and not to involve the great cities of New York and Brooklyn, where no yellow fever occurred. Then I am cognisant of the facts pertaining to numerous other epidemics, where a clear history of importation has been made out.

Governor's Island is entirely occupied for military purposes, and was thoroughly well policed, which could not be said at that time for New York and Brooklyn. Believing, then, as fully in the importation of the disease as if I could name the vessel which brought it, I am yet only able to offer you a theory in regard to it. The Atlantic Docks, Brooklyn, are directly opposite, and about 500 yards distant from that part of the island where the disease first made its appearance. Here it was ascertained that vessels coming from infected parts had discharged cargo a short time previously; but no cases of yellow fever were known to have occurred among the crews or labourers who had assisted in discharging them. Projecting from the island in the direction of the docks is a spit of land, which is uncovered at low tide, and which constantly has stranded upon it things which may be thrown into the water on the Brooklyn side. Just at the back of this spit of land is the house where the first cases occurred, and for a week the disease was confined to this house.

There were living in this house about a dozen persons (here I would say that the absence of my notes prevents me from being entirely exact in regard to dates and numbers). Of those living in the house, all but two, I think, were taken sick, and several died. A number of persons (thirteen, I think), who came to this house to visit friends before the nature of the disease was made out, were soon after taken sick in New York, and several of these died. From this house the disease extended itself along the shore in both directions, invading successively the hospital, the barracks of the band, and of the field musicians, and the laundresses' quarters on one side, and the officers' quarters on the other.

In the centre of the island, which has an area of 65 acres, is Fort Columbus, which at this time was occupied by three infantry companies. No cases occurred among the men quartered in Fort Columbus. On the north-eastern corner of the island is Castle William, an old fort, which is used as quarters for general prisoners. The occupants of this building escaped with few exceptions. The occupants of the Arsenal on the northern side of the island all escaped.

As soon as a decision was arrived at as to the nature of

the disease, all the sick were removed to the New York quarantine station, where I accompanied them as medical officer in charge. At the island vigorous measures of disinfection were put in force, but the great mistake was made of not removing all well persons from the infected area. The vain hope was indulged that sending away the sick was banishing the disease. The result was that new cases occurred daily, and were sent to me at the quarantine station, until, with hardly an exception, all who resided within the infected area indicated were stricken down with the disease. The whole number of cases was 150, of whom 50 died. As evidence that the disease was not propagated by personal contagion, I may state that at the quarantine hospital there were eleven persons who had never suffered from yellow fever, and that not one of them contracted the disease from the 150 yellow fever patients who occupied the same building with them. The bedding and clothing of these patients was all destroyed upon their arrival at the hospital. On the other hand, new cases occurred daily within the infected area at Governor's Island, notwithstanding the removal of the sick, and the burning of clothing and bedding which had been used by them. Again, those persons who went to New York after visiting the infected locality, and were there taken sick, did not communicate the disease to any one in that city. These cases were carefully watched by the New York health authorities, and disinfection was resorted to in the apartments occupied by them.

Having now given you the main points in regard to this epidemic, I must ask you to accompany me to Fort Barrancas, Florida, where, in the summer of 1873, I next encountered yellow fever.

Fort Barrancas is situated at the entrance to Pensacola Harbor on the mainland, and directly opposite, on the extremity of Santa Rosa Island, is Fort Pickens. The navy-yard and the village of Warrington are distant about a mile from Barrancas, and are separated from it by a cedar grove. The city of Pensacola is nine miles distant. The situation of Fort Barrancas is an exceedingly healthful one; the soil is sandy, and the natural drainage all that could be desired. The prevailing winds are from the south, and come fresh and pure from the Gulf of Mexico. On the north are extensive pine forests, noted for their salubrity; paludal fevers prevail to a very slight extent, and the cases are of a mild form.

During the year referred to yellow fever was introduced into Pensacola Harbor by the British ship *Golden Dream*,

which sailed last from Havana and came to Pensacola for lumber. This vessel came in with cases of yellow fever on board, and was at once sent to the quarantine station; here she remained until the last of her crew was dead or convalescent, when, in compliance with a ridiculous law, founded upon the idea that the disease is propagated by personal contagion, she was allowed to come to the city, as the legal time had elapsed since the occurrence of the last case. Soon after, cases of yellow fever occurred on shore, and a severe epidemic followed, which not only devastated the city of Pensacola, but extended to Montgomery, a city over 100 miles distant in the interior. Montgomery is connected with Pensacola by rail, and the disease germs were doubtless carried there by some of the many refugees who fled from the pestilence in the latter city.

As soon as we had information at Barrancas that yellow fever had appeared in Pensacola, a policy of absolute non-interference with the city was adopted, and most stringent measures taken for carrying it into effect. Our neighbours on the Navy Reserve adopted the same policy. About the same time that yellow fever was introduced into Pensacola Harbor by the *Golden Dream*, the *Valparaiso*, also from Havana, brought it to the city of New Orleans. This vessel had also complied with the quarantine regulations of the port. She had been detained at the quarantine station for a certain number of days, and, all on board remaining well, was permitted to come to the city. Upon her arrival the hatches were opened to take out the cargo, and the mate, who slept near them, was soon after taken sick with yellow fever. Others of the crew followed, and about the same time cases occurred on the *Belle Lee*, a river steamer lying next her. These facts I have from the Secretary of the Board of Health of New Orleans. Soon after, yellow fever made its appearance in Memphis, where an extremely malignant epidemic was rapidly developed. In New Orleans the disease made comparatively little progress, and this is ascribed by the health officers of this city to the vigorous processes of disinfection resorted to. Wherever a case occurred the clothing and bedding of the individual were disinfected or burned; the house in which he was taken sick was fumigated with sulphurous acid gas, and drenched with carbolic acid in the form of spray from a steam atomizer. The streets, alleys, and yards about every centre of infection were repeatedly sprinkled with a solution of carbolic acid, and, indeed, everything was done which science could suggest and energy execute, with a view to destroying the

materies morbi of the disease wherever it was supposed to exist. The small city of Pensacola, in which nothing of this kind was done, suffered much more severely from the disease than the large city of New Orleans.

For a time I flattered myself that the garrison of Fort Barrancas, as a result of our rigid quarantine regulations, would entirely escape invasion by the pestilence; but I was doomed to disappointment. Four cases of yellow fever occurred in the port hospital during the latter part of September, almost simultaneously. The patients were all men who had been for some time in the hospital under treatment for other diseases of a trifling nature.

My hospital was situated at a considerable distance from the company barracks and other buildings at the port. It was on the brow of a bluff some 30 feet high, and had an admirable outlook over the Bay of Pensacola and the Gulf of Mexico. There was no yellow fever nearer than Pensacola, and between that city and my hospital was, first, the garrison of Barrancas, next, the cedar grove, then the town of Warrington and the navy yard, and, finally, seven miles of pine woods. I could not understand it, but nevertheless there were the four cases of yellow fever which had originated in the hospital. I had the strongest reasons for believing that these men had had no communication with the infected city of Pensacola, and as patients in the hospital they had been under my daily observation. It was evident to me, therefore, that these men had contracted the disease on the spot, and that my hospital had become infected from some source. After a careful inquiry, the only theory I was able to form as to the mode of infection was the following. We occasionally purchased supplies for the hospital in the village of Warrington. Two weeks previously a barrel of potatoes had been so purchased; I ascertained from the merchant who sold them to us that they had just been landed from a New Orleans steamer when he received the order, and were sent directly from the steamer to the hospital. The barrel was there emptied by the steward on the floor of the storeroom. There were many rotten potatoes, which were sorted out and thrown away. Some of the potatoes were sent to the hospital matron, who lived in a small house situated thirty yards from the hospital storeroom. In this house two children, aged eight and ten, were taken sick with yellow fever, two days after the cases occurred in the hospital. These children both died, as did one of the men in the hospital. Now whether this barrel of potatoes was really the medium by which my hospital be-

came infected, it is, of course, impossible to say, but it seems to me not at all improbable that such was the case. There were three other barrels of potatoes landed from the steamer at the same time, and these were sold by the merchant to other customers without any bad results. You will remember that yellow fever had some time previously to this been brought to New Orleans by the *Valparaiso*, and carried to Memphis by the *Belle Lee*, or some other river steamer. This very barrel of potatoes may have been brought down the river on an infected vessel, or it may have been stored in an infected warehouse in New Orleans. The fact that the three other barrels conveyed no infection may have been due to their not containing rotten potatoes, or to their having been from a different lot before being shipped from New Orleans.

Immediately upon the appearance of yellow fever in my hospital the main portion of the garrison was removed into camp near Fort Pickens on the other side of the bay. All communication with infected localities being strictly guarded against, not a case occurred in this portion of the command. It was necessary, however, to retain a certain number of men at Barrancas, to take charge of the Government property, and, some of these men being married, their families also remained. Of those who remained, eleven were taken sick with yellow fever. None of these cases, however, came from my hospital, but all from a new centre of infection, which was subsequently developed in the company barracks.

I am convinced that yellow fever was stamped out in the port hospital by the measures which I immediately resorted to for the disinfection of the building. Every ward was repeatedly fumigated with sulphurous acid gas, and the floors, hallways, etc., were daily sprinkled with a strong solution of carbolic acid. In addition to the four men first taken sick there were in the hospital several patients (three, if I remember correctly), three attendants, and one steward. These all escaped the disease during the remainder of the season, although four additional cases were admitted from the company quarters. The beneficial results of removing the well persons from an infected locality, instead of the sick, immediately that proof of infection exists, will be apparent in comparing the result at Barrancas in 1873 with that at Governor's Island in 1871. The former place had the disadvantage of being in a much warmer latitude, and consequently was more favourably situated for the development of an epidemic; but under the policy adopted, but seventeen cases and six deaths occurred, and of these, eleven

cases were among those necessarily detained at the infected spot. The opposite policy adopted at Governor's Island resulted in 150 cases and 50 deaths.

In 1874, yellow fever was again introduced into Pensacola by a Spanish vessel, the *Doce de June*, which was sent to the quarantine station, but communication took place between the shore and the ship. Yellow fever soon broke out in a sailor's boarding house, and among the marines at the navy yard. The citizens of Montgomery escaped, through the observance of strict quarantine. The garrison of Fort Barrancas was moved into camp, and escaped with two exceptions, that of an officer who was in the habit of walking into Warrington, his attack occurring simultaneously with several others in the village. He was attended in his own quarters, and the disease did not spread, although his son slept with him at the time of the attack. The other case was that of a hospital attendant, who visited the village of Warrington without leave.

In 1875, the ship *Von Moltke*, from Havana, having cases of yellow fever on board, arrived in Pensacola harbour on the 28th of June. She was anchored by the pilot who brought her in, in the channel opposite Fort Barrancas. The next morning she proceeded to the quarantine station, and no communication was permitted with the shore. The result was perfectly successful, so far as the city of Pensacola and the navy yard were concerned. The entire crew of the *Von Moltke* suffered from yellow fever, and after the recovery of the survivors the vessel put to sea without a cargo, no communication having been permitted between her and the shore.

Three weeks after the arrival of the *Von Moltke* yellow fever again made its appearance at Barrancas, a number of cases occurring almost simultaneously in three different localities, viz., in the company barracks, in the laundress's quarters, and in the officers' quarters. A severe epidemic was developed with remarkable rapidity, as will be shown by the following figures. The first case was that of Mrs. Mary O'Halloran, a laundress aged 27, who was nursing an infant, and was in feeble health. She was taken sick on the 18th of July. On the 19th, Sergeant Correl, a married soldier, living in the same building, was taken sick. On the 20th, Mrs. Correl, wife of the above sergeant, and two enlisted men from the company barracks. On the 21st, five new cases, one an officer's child from quite another part of the garrison. On the 22nd fifteen new cases; and on the 23rd twelve, making thirty-seven in all on the fifth day

after the appearance of the first case. This was a different experience from any I had previously had, for it was evident the infection was not confined to one or two points, but that the whole garrison was pervaded by the poison. On the 21st the command was removed to Fort Pickens, but the removal was not in this instance immediately effectual in staying the progress of the epidemic. New cases occurred in camp for five days after the removal, and then there were no more. On the first day eight cases were sent back to Barrancas for treatment; on the second day twelve cases; on the third day seven cases; on the fourth day three cases; and on the fifth day two cases. This strongly confirms an observation I have made in numerous other instances, that the period of incubation is from two to five days. After the fifth day we received no more cases from the camp at Pickens, and the balance of the command remained there in safety for the rest of the season. At Barrancas, however, cases continued to occur among those who had been retained for duty, and in the families of some officers and soldiers who desired to remain in their quarters, until, in the end, every one in the garrison who had not previously suffered an attack of the disease was prostrated by it. I was myself one of the last victims, and had a very severe attack and tedious convalescence.

The total number of cases during the epidemic was 76, of whom 31 died. I have no doubt in my own mind that the origin of this epidemic must be traced to the *Von Moltke*, which anchored in the channel opposite our post three weeks before the occurrence of the first case. Shortly before this time there had been a severe gale, and a great deal of seaweed had been deposited upon the beach. A very unpleasant odour from the beach prevailed both before and during the epidemic. My own belief is that, in some way, yellow fever germs from the *Von Moltke* reached the beach—whether wafted by the wind, or floated upon infected articles thrown overboard, I cannot attempt to say—and that, finding there conditions favourable to its development, the poison multiplied itself so rapidly that, three weeks after, its fatal influence upon man was manifested by the epidemic which occurred.

This theory will doubtless appear unsatisfactory to some, and other ways of accounting for the outbreak will suggest themselves; but after a careful consideration of the facts, I have not been able to find any other which is satisfactory to my mind. That the disease did not originate at Barrancas *de novo* seems almost certain, for the following reasons.

Yellow fever has prevailed at Barrancas but six times in fifty-four years. Its appearance in every instance has been preceded by the arrival in the harbour of a vessel from an infected port.

The location of the post is ordinarily a very healthful one. The drainage is good, the water for drinking is pure, being obtained from cisterns, and the sanitary conditions generally all that could be desired, much better, certainly, than at Pensacola or Warrington, where in 1875 there was no yellow fever.

That the disease was not introduced by direct contagion from persons on the infected vessel we have the strongest evidence. In the first place, such a mode of communication is contrary to experience; secondly, a searching investigation failed to show that any communication took place between persons on the ship and any one at Barrancas.

We also have an affidavit from the captain of the ship that no intercourse with the shore occurred. Thirdly, the ordinary period of incubation in yellow fever is about five days, but it was three weeks after the vessel was sent to quarantine before the first case occurred. Fourthly, the first case was that of a woman who had recently given birth to a child, and had not been out of her room for a considerable time.

As to any other source of contagion, all that we can say is that, with the exception of the cases on the *Von Moltke*, yellow fever did not prevail anywhere on our coasts at the time of the outbreak at Barrancas.

But there is still another mode in which the disease may be supposed to have originated. May not the contagion have been preserved from a previous year in the bedding or clothing of a former patient? It will be remembered that but two cases occurred at Barrancas the year before, while a severe epidemic occurred at Pensacola and the navy yard, yet there was no recurrence of the disease at these places. And, again, the first cases were in a new building, the laundress's room, which had not been occupied until the spring of 1875, and among persons who, with all their clothing and effects, had been shut up in our harbour of safety at Fort Picken during the preceding years.

I do not wish it to be understood that I consider it impossible for yellow fever germs to retain their potency from one season to the next. On the contrary, we have the strongest evidence that such is sometimes the case. There are well authenticated examples of vessels upon which the disease has reappeared several seasons in succession, although

no infected part had been visited, except in the first instance. The disease has been known to reappear on vessels of our navy after they had been sent as far north as Portland Maine to pass the winter, for the express purpose of freezing the infection out of them. When you remember how great a portion of a vessel is below the water line, and consequently surrounded by a medium having a temperature above the freezing point, you will see that the freezing-out process is quite impracticable without docking a ship. At Barrancas, however, we have in the course of nearly every winter some pretty substantial frosts, and to these we are probably indebted for the fact that yellow fever is an exotic. Further south, where there is no frost, the germs hibernate, and the disease is indigenous.

The view which I have been forced to take of the nature of the yellow fever poison reconciles many facts which have heretofore appeared contradictory. If the poison is capable of self-multiplication, independently of the human body, we are not required to believe in the very prolonged period of incubation which has been recorded in certain cases. La Roche, in his work on yellow fever, gives one case in which the period of incubation was three months; and there are well authenticated instances in which the disease has first appeared on shipboard twenty days after leaving port.

Instead of attributing these cases to exposure before leaving port, and consequently looking upon them as examples of a prolonged period of incubation, I suppose the disease to have been contracted on shipboard, and that the infection was introduced before leaving port, but required a certain time for its increase before it was sufficient in amount to produce its characteristic effects upon the human system. I may here say that I have some reason for believing that to produce an attack of yellow fever it is necessary that the individual be exposed to the action of the poison in a certain degree of concentration, or, in other words, that a tolerably definite dose is required to produce the poisonous effects, and that other things being equal, such as individual susceptibility, which depends upon temperament, age, condition of health, etc., the severity of the attack depends upon the degree of concentration of the poison in the locality where the disease is contracted.

There are certain instances, cited by believers in the contagiousness of yellow fever, in which individuals have apparently contracted the disease directly from the clothing or bedding of persons sick with the disease. These cases prove that the clothing or bedding in question was infected, but

not that the infection was a result of it having been in use by one sick with yellow fever. The fact of the man having yellow fever proves that he had previously visited an infected locality; and, according to my view, the infection of his clothing was a result of this visit and not of his sickness. The most minute quantity of the poison conveyed about his person would be capable of indefinite multiplication if subjected to favourable conditions. Such conditions are found, I believe, in the sick bed of a yellow fever patient. The bed is commonly saturated with moisture, from the profuse perspiration which occurs during the first days of the disease. It is kept warm by the patient's body, and organic matter is given off by his skin and lungs in such quantity that the weight of his body is reduced many pounds in the course of a few days. If the yellow fever germ has been introduced into this bed it is not surprising that it becomes so thoroughly infected as to be a source of danger to all who come near it. It is for this reason that I always recommend the destruction or thorough disinfection of the clothing and bedding of yellow fever patients. If the views which I have advanced as to the nature of the yellow fever poison are correct, the means to be adopted for the prevention of epidemics are apparent, and it is exactly these means which modern sanitary science has successfully employed against this disease and others which in their etiology resemble it.

"COKO", A FIJIAN DISEASE.

By WM. MACGREGOR, M.A.,
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(Communicated by John Simon, Esq., D.C.L., F.R.S., Medical Officer to the Privy Council and Local Government Board.)

(*Read: June 14th*, 1876.)

[Abstract.]

In studying the nosology at this new colony the first disease that calls for special attention is "coko" (pronounced thoko), on account of its ubiquity over the entire country, its striking symptoms, and the conspicuous place it must have occupied among Fijian diseases before the appearance of measles and typhoid fever in these islands. Coko in the mouth of a Fijian is a generic term that is used to designate certain diseases of the skin that differ widely from each other. Two of these will be dealt with in this communication.

Amongst other names used by the Fijians to indicate different forms of the disease are "coko tanna" and "coko dina," under which I believe all forms of the disease are included. "Coko tanna" may conveniently first be disposed of. It is so called because it is said to have been introduced here from the Island of Tanna about ten years ago. I have seen about a dozen cases of this disease, and there is no doubt whatever in my mind that it is varicella.

This disease is very little, if at all, severer here than at home. I have never heard of a death from it. In all the cases that I have seen here the patients have been from about seven or eight to five and twenty years of age. An attack of it is no safeguard against an attack of "coko dina," and vice versâ.

"Coko dina" is, amongst the Fijians, a disease of childhood, usually attacking the child in its second or third year. Perhaps it is on account of its hoary antiquity and contagious nature that the Fijians have come to regard the disease as a necessity, for, instead of trying to keep their children free of it by segregation, they send them to live in

the immediate vicinity of affected children, if the disease has not appeared at the usual age, for no Fijian mother believes that her child can ever become a healthy adult without undergoing "coko". Just as with most other diseases, however, occasionally a child remains proof against the contagion, even when direct inoculation is practised.

For some time after exposure to the contagion the patient often suffers from feverishness and well-marked pains in the lower extremities, before the appearance of the specific eruption on the skin; but this is not invariably the case, else the fever must often be trifling, as several white persons have told me that they had no preliminary fever. Sometimes this incubation stage is prolonged and very severe, and, when it assumes this form, the disease is, on account of its mortality, greatly dreaded, as the child usually dies unless the eruption comes out. This form of the malady is called "coko e lomn", *i.e.*, internal coko.

After the incubation stage has lasted for some time the eruption begins to make its appearance. In those cases which I have had an opportunity of witnessing in this early stage, the eruption was first seen, as a rule, about the corners of the mouth, or about the fundament, apparently by preference always at or near the junction of the mucous membrane with the skin.

At first it might be mistaken for a commencement of herpes, but it soon appears as if a small soft condyloma was developing itself. It becomes slightly elevated, the epidermis separating from the cutis, and a small quantity of seropurulent matter gradually forming on the top of the latter, and giving the sore a peculiar whitish yellow colour. At the same time there is a circumscribed effusion of lymph going on into the cutis, which then eventually forms tubercles of greater or less size and elevation. Soon the sores appear indiscriminately over any part of the entire body, notably, as a rule, over the face, round the mouth, nostrils, and eyes. In a fully developed case I have counted over a hundred sores at one time, and there are usually fifteen or twenty of these about the face and neck. The sores are usually of a circular form or elongated with rounded corners, varying in diameter from two inches downwards, and elevated above the surrounding healthy skin to a varying degree, sometimes as much as half an inch. Occasionally the tubercle is seen ring-shaped, with a spot of unaffected skin in the centre, and sometimes by the coalescence of several contiguous tubercles an irregular looking sore may be produced, but individual sores appear to always retain the rounded form.

From the surface of some of the tubercles or sores a slight discharge of purulent matter takes place, mixed up with serum and disintegrated epidermis, forming a thin covering of a creamy colour over the summit of the tubercle. This crust is easily removed, usually coming off entire, like a cap, from the top of the tubercle; beneath it there is a small quantity of purulent matter, through which protrude what appear to be papillæ of the true skin. They are very much enlarged, standing up separately, sometimes one-eighth of an inch in length, the brightness of their hue appearing a strong contrast to the creamy fluid bathing their almost contiguous bases. Left to themselves, it is rather rare for these sores to cause destruction of the cutis and permanent cicatrices. They often disappear, even when of the largest size, in a week or two; the lymph effused into the tubercles is absorbed, and the tubercle subsides; the enlarged papillæ disappear, and the affected spot attains the level of the surrounding skin, leaving merely an evanescent discoloration of the epidermis.

It is usual for several crops of tubercles to succeed each other for a varying length of time before complete recovery takes place.

While writing this I have been consulted by a white family, the mother and two children of which have suffered from this disease for about two years; but that, I believe, is considerably over the average duration, which may be stated as being about twelve months.

During the course of the disease there generally, if not always, appears one sore that is conspicuous among the others from its greater size, its longer duration, and often from its structure. It frequently causes destruction of the true skin, and degenerates into a circular depressed ulcer, which, after attaining a diameter of about two inches, remains stationary, until the tubercles are finally disappearing. This is its usual character when situated below the knee, but if located near the fundament it presents a different appearance.

In two cases under treatment at present this sore presents the aspect of a soft, flat, warty condyloma, about one and a half inch in diameter, and elevated about one sixth of an inch above the level of the surrounding skin, close to one side of the anus. This peculiar sore is called the *vu ni coko*, (root of coko), or *tina ni coko* (mother of coko), because it is often the first to form, and usually the last to disappear.

There are several peculiarities connected with this eruption that remind one of certain exanthemata. Thus, the

universal opinion here is that it is communicable by inoculation, and the common house-fly is credited with this operation. Recently I had an opportunity of inoculating an adult Polynesian coko patient at various times with different varieties of the discharge from his own tubercles, but at none of the inoculated spots did a tubercle or sore form.

The disease is too serious to warrant the inoculation of healthy persons that have not previously suffered from it, so that I have no positive proof in my own experience that it can be inoculated; but the above experiments, taken in connection with the fact that the relative positions of the tubercles indicate that the occurrence of a sore at any particular spot is accidental, and that one tubercle does not arise at any particular place from matter having reached that place from a pre-existing tubercle, would lead to the inference that the poison is constitutional, as is also shown in the whole history of the disease.

Another point of resemblance between coko and the exanthemata is that the eruption sometimes does not come out at all, or if it comes out it recedes, or is driven in by cold, or by the occurrence of some other disease, and these cases are always regarded as serious, especially the latter. I have been told of many cases in which the eruption dried up during the epidemic of measles, when the patient was attacked by the latter disease. Sometimes it came out again after the recovery of the child from measles, but occasionally it did not reappear, and the latter class of cases is believed to have been, without exception, fatal. I have seen one case, and I am told it is not unique, where the eruption disappeared, and the patient seemed to enjoy fair health for about three months, but after that time the eruption reappeared, and the same patient has been undergoing treatment for the relapse during the last two months. There are examples of a patient undergoing a second or even a third attack, but these occurrences are rare. There is a daughter of a chief on this island who is said to have had the disease five times, but, if that is true, it must be a very exceptional case. I have been informed that children have been born suffering from the disease, and that the child so born always dies, but I have never seen an example, as it must be very uncommon, and, indeed, can scarcely occur among Fijians, but might happen among Europeans, or imported labourers. It would appear that "coka dina" is not common to all the Polynesian islands, because the foreign labourers working there very frequently contract the disease, and they tell me they never saw it before their arrival in

Fiji. Nothing can well be more annoying European than to suffer from this loathsome disease for a year or two. Fortunately, i among them is the exception, and not the believe with ordinary care may always be avo

Treatment.—The family of three I alluded been treated by Fijians for upwards of two yea native treatment is not very encouraging i Their usual plan seems to be to take the chil shore, and with a piece of cloth to scrape ea tubercle until quite raw, and bleeding freely, the tubercles have been scraped, to plunge peatedly into the salt water; or, instead of th ceeding, after the scraping they squeeze green the sores.

This treatment is, I believe, put in force onl stages of the disease, when the Fijian mother her child has had a sufficiency of it. It is s heal the existing tubercles, but that new ones while. Lately there were two cases in the pu for treatment. Each patient, father and son, L had suffered from the disease for about one each when admitted were thirty to forty tuber one case the crusts were ordered to be remov sores, and mercurial ointment applied daily. case no treatment was applied beyond drawin nitrate of silver round each tubercle to mark as I was about to leave hospital on official busi or twelve days. I also removed the crust from largest of his tubercles, and applied the solid l freely to the papillæ to see the effect on my first seeing these two patients, twelve days quite astonished to find both had completely re that the only trace left by each tubercle was tion of the epidermis.

But these two cases only illustrate the amena disease to treatment in its later stages, for it is mon to treat less chronic cases for months withc a complete cure. Indeed, in the two cases first cure was probably due to good food and lod experience is wanted, and a close observance of pathological relations of the disease, before ar line of treatment can be recommended.

ON DENGUE, OR DANDY FEVER.

By Sir W. R. E. SMART, K.C.B., M.D., R.N.

(*Read: December 13th*, 1876.)

[Abstract.]

Part I.

In June 1865, I read before this society a paper on dengue, having witnessed two epidemics of it at Bermuda. (*Trans.*, vol. ii, part ii.) Since that time it has attracted great interest by its wide epidemic extension over the coasts of the Indian Ocean and its gulfs, from Suez to Zanzibar, and to Java in 1871; from Aden to India, Burmah, and the Malay Peninsula in 1872; and from Singapore to China in 1873. In each locality it has incited able recorders of its phenomena. Among these I would mention Vanoray in Suez and Egypt; Read in Aden and Arabia; Christie in Zanzibar and East Africa; Da Cunha in Bombay; Moodeen Shereeff, and Chipperfield in Madras; Slaughter in Burmah; Dr. Charles in Calcutta and Bengal; De Wilde in Batavia and Java; Müller and Manson in Amoy and China.

These authorities have afforded proofs of the identity of the disease on the eastern with that of the western hemisphere, and its history extends to the same period on the coasts of the Atlantic and Indian Oceans. Its limits have been tropical or sub-tropical, or where a high summer heat is maintained. Within the tropical zone it has been an epidemic from Peru to Senegal, and from Zanzibar to Tahiti, and north of that mildly epidemic in Bermuda and New York, Spain, Egypt, Northern India, and Amoy, in China, also in the Sandwich Islands, and south of it, in Brazil and in Bourbon. In both hemispheres its range is almost identical with that of so-called bilious remittent fevers, and in the western hemisphere with that of yellow fever, but not so in elevation, since it has been epidemic in the uplands of Mexico, to which yellow fever does not ascend; and it was seen by Poggio at Orotava, in Teneriffe, 1,200 ft. above the sea level. Of the various recorded diffusions of this fever I give the distinction of "pandemic" to

those periods in which the disease has existed in both hemispheres, and that of "epidemic" to those in which it has been observed only in one hemisphere.

The first on record extended from 1778 to 1784. This includes the mixed fever that attacked the inhabitants of Philadelphia after the evacuation of that city by the British troops, which has been described as bilious, yellow, or remittent fever (Bascome's *History of Epidemic Pestilences*, p. 136), followed, in the summer of 1780, in Philadelphia by "break-bone fever, so called in consequence of the violence of its attendant pains"—(Rush's *Medical Inquirer and Observer*, Philadelphia, 1789); the sequel of the mixed fever of 1778, as later it was of that in the Southern United States in 1849-50. This terminated the epidemic in the western hemisphere.

Within this period it was recorded in 1779 in the eastern hemisphere, at Batavia, by David Brylon, the chief colonial physician, who described it as *knockel-koorts* (bone fever). (J. J. de Wilde, *Niedr Tjidschr.* 1873.) In 1780 a missionary, Wise (according to an anonymous French writer), observed the same disease on the coast of Coromandel, Egypt, Africa, and Arabia, and it was described in Spain by Cubilal and Nieta in 1784. It was a pandemic from 1778 to 1784. Its manifestations from the last date till 1824 were regional epidemics only, so far as it is recorded.

The second pandemic, from 1824 to 1828, commenced in the eastern hemisphere, at Calcutta, in June 1824, and travelled along the banks of the Ganges to Berhampore in April 1825. It was well described by the English physicians, Mellis, Kennedy, Twining, and Mouat, in the *Transactions* of the Medical and Physical Society of Calcutta for 1824 and 1825. It was defined by Mellis as "epidemic inflammatory fever", and by other physicians as "eruptive joint fever", now "eruptive rheumatic fever"; and, as in Philadelphia it was known popularly as break-bone fever, so in Calcutta it was called red fever and three days' fever, expressive of its most prominent and obvious features through the Bengal and Madras Presidencies.

In the western hemisphere the first appearance, according to Waring, was at Savannah, in the autumn of 1826, but its great diffusion took place from the Island of St. Thomas, in the Virgin group, where it commenced late in 1827, and was transmitted through the Carribean Islands to leeward, at the season of the year when north-east winds facilitated communications with the south, so that after reaching Barbadoes it passed westward to the Spanish main and its islands early in 1828.

According to Dr. Wood "it proceeded from St. Thomas to the Continent, reaching New Orleans in the spring, and Charleston and Savannah in the summer and autumn of 1828," when it prevailed also at the Bermudas. There is no proof of its having extended in epidemic type to any more northern latitude. During this diffusion it received the name of "dandy fever" from the negroes of St. Thomas, which was translated into *dengue* in the Spanish tongue, both meaning the stiff carriage of those affected by it, now its accepted appellations among all Teutonic and Latin nations.

This second pandemic lasted from 1824 in Calcutta to 1828 in the United States. It is noticeable that an approximate term of forty years elapsed between these two so-called pandemic cycles, and it is equally remarkable that nearly an equal interval stands between the last of them and that of recent occurrence.

The third pandemic period, which has presented none but sporadic cases in the West Indies, has extended from the head of the Red Sea to China, and from Zanzibar to Java, having shown itself in the summer of 1871 at Port Said, Aden, and Zanzibar, possibly disseminated by Mahommedan pilgrims and traders. Moodeen Shereeff, who has so well described the epidemic in Madras, states, from personal knowledge, that it was in Arabia for several years previous to 1870, and that in 1871 it prevailed at Mecca, Medina, Tanjef, and Jedda. Dr. Reid of the Rifles, stationed at Aden, has described its appearance there, and Dr. Christie, physician to the Sultan of Zanzibar, has detailed its epidemic characteristics on the east coast of Africa. He states that it prevailed epidemically in Zanzibar from July 1870 till January 1871, and he does not afford any information as to its introduction from without. Dr. Reid states that it broke out epidemically in Aden in June 1871, and that it was brought from Zanzibar by trading vessels.

There is the interval of an entire year between July 1870, when it broke out epidemically at Zanzibar, and June 1871, when it did so at Aden, and as southerly winds of the southwest monsoon prevail in the Arabian Gulf between April and October, including two months immediately preceding the date of its outbreak at Aden, none but sailing vessels having intercourse between Zanzibar and Aden, it is highly probable that Aden was directly infected from Zanzibar. Although it may be an open question whether it was imported into the Hed-jaz from Aden or from Zanzibar, it is noteworthy that before taking any diffusion by longitude it

covered 38 deg. of latitude, from Zanzibar to Port Said, in the summer of 1871. Its transmission from Aden to India by the steam trooper *Dalhousie*, in December 1871, is unquestioned; its diffusion over India in 1872, and from it into Burmah and Pegu, and the Straits settlements, and thence to China, and possibly into Java, if not taken there direct from Djeddah, is now a matter of history.

It will be objected that, as there has been during this period no great outburst in the western hemisphere, this would be scarcely a pandemic in accordance with the definition with which I set out. In answer I may refer to the fact that our Navy Returns for 1870, the year in which it appeared in Zanzibar, show that cases of it were noted in ships of war—the *Aboukir* and *Defence*—at Jamaica, and yellow fever had been epidemic in that station, and strongly so in those two ships, in the preceding year 1869, and more mildly displayed in the *Aboukir* in 1871. In fact, yellow fever was felt severely on the West Indian Station from 1869 till 1873, embracing the term of the dengue pandemic of the East, where yellow fever is unknown; but in the West, dengue has been found on several occasions to represent the milder type in epidemics of yellow fever.

Having detailed the principal facts relative to the three historical greater epidemics or pandemics, let me now summarise the regional epidemics that have been noticed in the long interval of about forty years between these pandemic periods.

Between the first and second, after its subsidence in tropical regions, it appeared in the Peninsula, the only European region in which yellow fever has ever become epidemic. Spain was ravaged by epidemic fevers from 1780 to 1787, and during this period, in 1784, and when virulence had ceased to characterise the prevailing disorders in 1788, this peculiarly mild form took possession of the community of Cadiz, on which account in its after-spread it obtained the name of the "Cadiz-fever" elsewhere in Andalusia.

By Don Christoval Cubilal its Cadiz name is given La piadosa, or the merciful, by Ferdinand de Castilla, who wrote a treatise on it in 1789, as influenza piadosa (vulgarly pantomima), and Nieto de Pina, who wrote concerning its visit to Seville in 1785, entitled his work *A History of the benign Fevers that prevailed in Seville from the beginning of September to the end of November*, 1784.

These references by Dr. Poggio display well the most obvious characters of the disease. It was epidemic in Spain

from 1784 to 1788, and there is no notice of its reappearance there before 1867, when an epidemic of it prevailed from the end of June, declining at the middle of October, as detailed by Poggio. In that interval yellow fever appeared on the rock of Gibraltar so fiercely that out of 14,000 souls only twenty-eight persons escaped, of whom twelve had previously suffered elsewhere (Bascome, p. 147).

When dengue was diffuse elsewhere in 1828, yellow fever decimated the garrison of Gibraltar, but it has not appeared there since that year. In Africa, in the first long interval, it was seen epidemically by Gabertz in Cairo in 1799, but it is said to have prevailed there also in the first pandemic in 1779.

The rarity of notices of the disease in the first interval may be attributed to its minor degree of importance where yellow fever or plague, and, lastly, Asiatic cholera, became indigenous. But since the second pandemic diffusion, from 1824 to 1828, greater attention has been devoted to its outbreaks. Thus, in its Indian region it was noted at Calcutta in 1836 by Dr. Raleigh; in 1844 by Dr. H. Goodeve; in 1847 Cawnpore by Dr. E. Goodeve; in 1853 in Calcutta by the same; and in 1854 by Dr. Mackinnon, and other notices of it have appeared from time to time in the Indian journals. All of these would appear to have been merely local epidemics, and it has been asserted by Dr. Charles that the disease is endemic in Calcutta, and that scarcely a year passes in which cases of it do not present themselves sporadically there.

Its presence in Egypt, as knee-evil epidemic, was noted by Prunek, in 1835, and again in 1845.

In the American region it has been recorded in Louisiana in 1831; Bermuda in 1837, concomitant with yellow fever; Herville in 1839; Louisiana and Mississippi in 1844; Brazil, in 1846 to 1849, followed by yellow fever; New Orleans, Mobile, and adjacent region, simultaneously with yellow fever in 1848; in isolated cases the next two years, until it became again epidemic in 1850 over the West Indies, and extended from the Atlantic shore to Texas; in 1852 and 1853 in Peru, being slightly mixed with yellow fever in 1853, which disease occupied the whole epidemic field in 1854 (A. Smith); at the Havana and Curaçoa in 1854 (Arbollya); at Bermuda and Martinique in 1860 (Ballot); in the West Indies and Southern United States, with yellow fever, in 1861; again in 1862, and in 1863, when it extended to Bermuda, where yellow fever committed great ravages the following year, 1864, the early cases being undistinguishable from

yellow fever; and, lastly, over the Southern United States in 1866.

On the eastern side of the Atlantic it appeared in Teneriffe in June 1865, and was there attributed to vegetable decomposition; but in the same year it prevailed epidemically throughout Senegal, having been previously epidemic there in 1845 and 1848, and at Goru and St. Louis in 1856.

In the South Indian Ocean it is reported by Dauvin to have been seen about this period, almost annually, in Bourbon, under the name of Chinese fever, and, medically, "the red epidemic fever", yet there is no report of its having been noticed in Mauritius, where at that time bilious remittent fever and dysentery filled up the epidemic field in sanitary reports. French naval physicians have noted its existence in the Polynesian groups from 1847 to 1856, few years passing without an epidemic in Tahiti, where it is styled sometimes "bilious fever".

In the Sandwich Islands, the disease known as "Bouhou", or the "groaning disease", is considered by French naval physicians to be no other than dengue, which began in Zanzibar, was conveyed to Arabia, and from thence to India, and so far east as China.

The recent great epidemic, if not previously existing on the continent of Africa, opposite to that island, originated in Zanzibar in July—the season of heat and moisture—1870, and continued there in epidemic form till January 1871. Most probably it was conveyed by sailing vessels that take the northern voyage after April to Aden, where it first attacked the natives, and became epidemic among the European troops in July and August. After the epidemic type had ceased, it remained amidst the natives in sporadic type, and in April 1872 it again became epidemic in the military quarters, more severely than in 1871, the scarlet rash being less pronounced and with a greater frequency of cerebral symptoms, which were attributed to insolation.

Late in the year it was carried from Aden to Bombay by native troops in the *Dalhousie* steam trooper, and during the first three months of 1872 sporadic cases were reported amongst the native population of Bombay in August (Da Cunha), but did not appear among the European troops before April, when, early in the month, it became epidemic, and lasted in that type up to the end of June, when it began to decline rapidly. Bombay being a city of great intercourse with the coast, as with the interior, both commercially and by the movements of regiments, it was diffused from it in every direction.

Thus it was conveyed among troops to Cannanore, far south of it, in the *Dalhousie*—the steam trooper by which it had been imported from Aden. In consequence of that circumstance she had undergone cleansing and painting, without use of disinfectants, while at Bombay, and then received European troops on board for Cannanore at the end of December. During the voyage her European officers and portion of the crew suffered from the disease, and immediately after their disembarkation at Cannanore the troops had an epidemic display amongst them, the first case occurring on the 2nd of January 1872.

From Bombay it spread to Poonah, by railway transit, and prevailed epidemically amongst the European troops, appearing in one regiment on the 10th of March, and in others on the 23rd and 24th respectively.

Kirkee, another military station, suffered about the same time. The epidemic reached its climax in April, after which it gradually fell off, and disappeared at the end of November.

From Poona it was traced directly with shifting troops into Madras, where it appeared epidemically towards the end of April in the forts of St. George and St. Thomas's Mount about the same time, but according to Dr. Chipperfield, the first case was noticed in Madras on the 8th of February 1872. It was fiercely epidemic from August till the middle of October, when, after heavy and continuous rains, at the change of the monsoon it became suddenly mild and sporadic. Possibly it may have been introduced in the cool season across the Peninsula by railway from Cannanore, and was reinforced later from Poona, when it took an epidemic type. Moodeen Shereeff, surgeon of the native hospital at Madras, states that he did not see any case until July, between which and the end of 1872 he had 600 cases under his observation. The disease did not cease in Madras Presidency throughout the year 1873, as the Army Reports instruct us, Trichinopoly and Kamptee being the latest infected. It was observed that April and May were the zero months of infection, showing only two and one attacks respectively, and that the greatest degree of it was in August. The slowness of decline in Southern India, where the temperature does not greatly vary, as in the northern provinces, is shown in the total attacks in the Presidency among the troops being 73 in January, and only 8 in December. In the Madras Presidency it appears to have lasted nearly two years, epidemically or sporadically.

Later in the season of 1872 it was taken, in a south south-east direction from Madras, when the epidemic was at its height there, by a detachment of infected troops, to Trichinopoly. There were no deaths from it among the troops in the Presidency.

Returning to Bombay, and thence tracing the disease northward, it is found early in June at Ahmedabad on the Sabatmattee, which falls into the Gulf of Bombay, and at Deesa, on the Bannas, that empties itself into the Gulf of Cutch, causing much alarm and suffering in both.

At Nupeerabad, in Rajpootana, north-east of the two last towns, it was not seen till November, when the cool season had quite set in; and likewise at Kurrachee and Hydrabad, on the Indus, it did not appear until September, becoming epidemic from October to December. In these three towns it was remarkable for its mildness in form as well as for its less frequency of attack, compared with those places in which the epidemic fell in the hot season.

We read the accounts of what happened in the north-west of India in the *Army Report* for Bengal, but we must not embrace the idea that epidemics in India answer to the roll-call of the army. Rangoon lies in the Madras Presidency, or command, but its epidemic of dengue is retraced to Calcutta, the great channel of its commercial intercourse; and likewise the central and north-west provinces are within the command of Calcutta, but their epidemic of dengue is most likely to have reached Benares and Dinapoor from Bombay, its port of arrival in India, than by a retrograde track from Calcutta.

The epidemic having fixed itself in Benares, travelled north-west to Allahabad and Cawnpore in the second quarter of 1872, and also to Lucknow in Oude, from Cawnpore. In the next quarter, the third, it extended further north-west by the Jumna to Agra, and thence to Delhi, Meerut, and Muttra, and lastly to Umballah, at the foot of the Himalayas, in the last quarter of 1872, having taken up a year in traversing the plains of India from Bombay to Umballah, being about the same time as for its diffusion from Bombay to Trichinopoly in the south-east of the Peninsula, through Poona and Madras.

In Calcutta, according to Dr. Charles, the first cases were in the autumn, that is at the end of 1871, and in a few months the disease spread over the whole city, reaching its climax in March 1872. He asserts its endemicity there, and that few summers pass without presenting sporadic cases of this fever. If this be the case, it remains to ascertain and define what are the local or the cosmic conditions under which this

so-called *endemic* disease assumes contagious or infectious properties and becomes *epidemic*. Does this disease, in its sporadic type, decline in contagiousness and require to be sown again, like annual flowering plants, with seed from other ground, to cause it to flourish with contagious properties, and to render it capable of epidemic diffusion? However this may be, it would appear that dengue was first seen at Calcutta in November 1871, in the poorest and dirtiest parts of the city—in the Jews' quarter, this people having much intercourse with Aden by direct steamers, as well as *viâ* Bombay. Now, as the disease appeared there as soon as, if not earlier than in Bombay, it may have been imported direct, and not through Bombay. By whatever channel the germs were imported, the first cases would seem to have been isolated in the winter months and not to have attained epidemic force among Europeans before March. From Calcutta it was conveyed by passengers on steamers to Rangoon, in Burmah, which military station lies within the Madras Presidency, but the source of importation of the disease may be rightly referred back to Calcutta, as cases were known to have occurred on board steamers from that port during the early part of the epidemic, previous to its appearance at Rangoon.

Surgeon-Major Slaughter, of the 10th Regt., has afforded us an excellent account of the epidemic there. He states that it made its appearance on the 15th of April 1872, and prevailed till the middle of July, and that during that time it was so generally diffused through the town, that few escaped, its contagiousness being very manifest. He has given thermo-track-charts, which display most graphically the sudden fall, within forty-eight hours of attack, and the remittency maintained throughout.

We possess three military stations in Burmah, viz., Rangoon, at the head of an estuary forming the harbour of Prome, on the Irawaddy; Tonghoo, 100 miles inland, on the river Sittang, and Thayetmyo, on the Pegro frontier. The disease was epidemic at Rangoon from April till August; in Thayetmyo from May to August, and at Tonghoo in August and September, with a few cases after that to the end of the year; but it revived in 1873, so far as to show 106 admissions, and lingered on through 1874 in disconnected cases. From Burmah it is said to have spread into Siam, but there are no records of this. It is to be regretted that the reports of our Colonial surgeons to the Colonial office are not made public, as much valuable information could be derived thence. It was not seen in

Penang. I have no knowledge of the disease in Singapore, the entrepot of the Archipelago, other than what is incidentally said by Muller and Manson concerning the epidemic in Amoy, that it had been imported from the Straits Settlements, where it had prevailed many months prior to August 1872, when it broke out in Amoy.

Whether Singapore became infected from Calcutta or from Arabia it may be difficult to ascertain now. It is the central point of departure and of return for the Mahommedan pilgrims of the Malay archipelago. There is no evidence to guide, but we may rest assured that any epidemic disease once fixed in it will soon be dispersed eastward and north-eastward. Thus it is said by Drs. Manson and Muller to have been fetched from that source to Amoy, where it prevailed extensively in the autumn of 1872, presenting then its normal phenomena with severity. How far it extended into China is quite unknown, but it certainly lingered long on the coast, as it was still prevalent, but not extensively so, at Hong-Kong, far south of Amoy, so late as November 1874.

As for the Malay Archipelago, we know nothing concerning it beyond that, according to Dr. Wilde, it was epidemic among the Dutch troops in Fort William, Batavia, in November 1871, about the same period as it was introduced into Calcutta, according to Dr. Charles.

Beyond these points of time and places of the diffusion of the epidemic that began in Zanzibar in July 1870 and was seen at Hong-Kong in November 1874, we are without further information, and here I must quit the tracing of its history, which has afforded ample proofs of the contagiousness of the disease and of its conveyance along the channels of human intercourse by direct contagion.

Part II.

(*Read: February 14th*, 1877.)

Few diseases have more numerous synonyms than dengue, owing chiefly to local or regional variations in the predominance of one or other of its phenomena. Thus groups of synonyms have arisen having relations to the altered state of the skin, or to the painful symptoms, or to the equal display of these, or to a first-sight resemblance to other diseases; and to either of these groups or single names the feature of epidemicity may have been appended or not.

The title now generally accepted is that given by the West Indian negro slaves to it when it first appeared among them in 1827—the Dandy Fever—from the strained attitudes of its sufferers, translated Dengue in Spanish, altered into "Denguis" for scientific classification.

The invasion of this disease is not usually attended by rigor, although the first stages of heat and sweating have been observed in the West Indies to have had throughout a sense of cold in painful degree. This stage may last from six to thirty-six or to forty-eight hours, during which the whole surface is of a more or less scarlet hue, and there are more or less acute pains in the head, spine, and limbs, creating much alarm. It often terminates abruptly, on awaking, in apyrexia so complete that the sick take to their ordinary avocations, a general stiffness and soreness in motion alone remaining, which gradually wears away in many cases; and to this condition the term dandy-fever was applied. But more frequently, after a period of from one to four days, there comes a second accession of fever, characterised by a rubeoloid eruption, which ends by desquamation and by a return of the pains, with which there is a remarkable fall of the pulse, sometimes to fifty beats. The continuance of this stage is uncertain, lasting but three or four days, then disappearing gradually, but sometimes very slowly, through weeks in which there are exacerbations that impart to the disease a relapsing character. The pathological features are those of an exanthem with muscular and arthrodial pains, without effusions into the joints, and with lesions of innervation, with diminished force of circulation, and gastro-hepatic disturbance, which may be indefinitely prolonged. In children the attack may be ushered in with convulsions, and the resolution may be attended by sensorial depression approaching more or less to "amentia"; this last condition may attend the stage of resolution of the disease in elderly persons also, but its duration is only temporary and concurrent with the induced conditions of general debility; nor can the disease, however severe, be said to have any special sequelæ proper to itself, like the typical exanthematous, which it simulates in its eruptions, or like acute rheumatism, to which it approaches in its painful symptoms.

Nosologically, its position appears to be with exanthematous fevers, while from all of these it is differentiated by the absence of uniformity of symptoms and normal course, of destructive organic lesions, and of definite sequelæ, by its liability to occur more than once in the same individual,

and by its extreme rarity of fatal results relatively to the amount of suffering it inflicts.

The infectiousness of "denguis" has been a disputed point from the moment when its epidemic nature first attracted attention. The affirmative opinion seems to have been first entertained in the West Indian epidemic of 1827, when it was traced from island to island by isolated importation. But even then there were those who attributed its diffusion to a widely spread so-called "epidemic constitution of the atmosphere", such as was at the same period maintained to be the sole cause of epidemics of yellow fever.

Now there are, probably, in that region very few acquainted with the disease, who doubt its intrinsic infectiousness by importation, or its propagation and diffusion from solitary cases, for this property of disease is more concisely defined in insular than in continental positions. And thus it has happened that the discussion has remained an open one to a later date in the East Indies. There, however, the facts connected with the last epidemic, 1871 to 1874, have brought the question within more definite limits.

The earliest epidemic display was at Zanzibar in 1870, and then at Aden, holding monsoon commerce with the former town in June 1871, both Zanzibar and Aden having commercial intercourse with India, especially with Bombay; and Zanzibar having such intercourse with the Arab coast of the Persian Gulf. It is therefore of interest that it was epidemic at Aden and at Muscat before any notice was taken of its presence in either of the presidencies of India. But, after it had become epidemic in Bombay and Calcutta in the spring of 1872, recollections arose of its having presented cases in Bombay so early as August, and in Calcutta as November 1871, both of these ports maintaining frequent intercourse by steamers and sailing vessels, some of them native craft, with Aden; and it has been stated by Dr. Charles—no mean authority—that cases of the disease are seen by him in Calcutta almost every summer. The period between the unrecorded isolated cases and the epidemic outburst having been such as to raise a doubt whether the entire visitation was not of Indian origin independent of importation.

It is clear, however, that there was no epidemic outburst in India previous to the arrival of a vessel from Aden, bearing the disease in epidemic form. This vessel was the *Dalhousie*, a Government steam trooper, whose movements were easily ascertainable. She arrived at Bombay from Aden early in December, then having the disease among

her Lascar crew, and on that account she was cleared out, cleansed, and painted, but not disinfected. After that she embarked European artillery, with whom she reached Cannanore, on the Malabar coast, southward, about the 1st of January 1872. During the voyage the disease broke out amongst her European officers, who had been in her from Aden to Bombay without suffering, when the native crew were attacked: and the troops she brought from Bombay only showed the epidemic type almost immediately subsequent to their landing at Cannanore, and the disease was afterwards seen as an epidemic in that district so far south as Alleppy and Cochin.

After the disease had established itself in epidemic type at Bombay and in Calcutta, it spread throughout India to the foot of the Himalayas, and from Kurrachee in the extreme west, to the banks of the Brahmapootra in the extreme east, extending along the railway routes, and being frequently conveyed from station to station amidst infected detachments of troops. It was thus carried into our garrisons in Burmah. It appeared in Singapore, from which place it was taken to Amoy, in China, spreading into the southern provinces. It also made its appearance in Batavia. In the whole of this wide dissemination there was sufficient evidence of its infectious, if not of its contagious, properties.

In the western hemisphere there has been a very observable complicity of its epidemics with those of yellow fever on several occasions, both as prodromic and as concomitant. This feature has been noticed in the Southern United States and in Bermuda, and it is further remarkable that the only European region it has yet visited is the Spanish Peninsula, which alone has undergone epidemics of yellow fever. The widest, however, of those connected diffusions was in South America, from 1846 to 1858. It appeared first in Brazil in 1846, which holds much commercial intercourse with the United States, where it had prevailed in Louisiana in 1844, and simultaneously with yellow fever in the summer of 1848 in New Orleans, and at Mobile in Alabama. It spread in Brazil during its summers up to that of 1849, and in the following year, 1850, yellow fever superseded it, recurring through six successive summers.

In Peru it commenced in January 1852, having broken out at Lima amidst a body of 1,000 German emigrants, who had recently arrived, after touching at Rio Janeiro, where yellow fever was epidemic. From Lima the disease spread far in the coast towns, and ascended to the inter-Cordillera provinces, but ceasing at Lima in July. Dr. Archibald

Smith has described it as a fever commencing with flushed face, injected eyes, and bruised sensations in the limbs and loins, ceasing about the fourth or fifth day by crisis of sweating, sometimes of hæmorrhages, with appearance of a roseolous eruption, the transition being often sudden from pyrexia to apyrexia; this eruption then lasting several days, often with spongy bleeding gums. The usual course was one of about eight days, leaving recurrent neuralgic pains for weeks or months, the fatal cases being few.

It disappeared from Lima in July, but reappeared in January 1853, attacking many of its previous sufferers, and generally unattended by the roseolous eruption. At the middle of March, Dr. Smith attended the first case seen of "black vomit, with jaundice", such cases being rare that summer. In January 1854, yellow fever of a very fatal type ravaged Callao and Lima, continuing through the summer, from which the attacks of the milder form in the previous summers afforded no immunity. It ceased on the advent of the cool season, and was unseen there in 1855. In the following summers till 1857-58, this epidemic made successive outbreaks on different sections of the Andes, in which climatic conditions seemed to modify greatly its severity. Thus in the dry province of Pinra, next the equator, it did not reach the fatal climax of black vomit; and, although hundreds of cases were introduced into the city of Arequipa, in the midst of a dry volcanic oasis, no new cases were known to have sprung from them; higher up the mountain range, however, at Cuzco, 300 feet above the sea level, it proved to be of the most fatal type.

This may be referred to by some as the most striking example on record of a long epidemic of yellow fever, which, in its first season, was undistinguishable from severe "dengue", and this modified character was attached also to its visitations in some of the mountain districts, but not so in all of them. As it was proved in Lima and Callao and elsewhere that attacks of the milder type afforded no immunity, as true yellow fever does, from future attacks, these diseases ought perhaps to be differentiated as distinct entities constituting one epidemic, the principal feature in common being the tendency to a shorter or longer period of apyrexia between the primary and the secondary stages of the febrile action. The question of the corelation of diseases in epidemic seasons or cycles, of which this seems to have been an example, and which is often observable in epidemics of other zymotic diseases, may be worthy of consideration by the Society.

ON MEASLES IN FIJI.

By WILLIAM SQUIRE, M.D., M.R.C.P.

(*Read: January 10th*, 1877.)

On January 12, 1875, soon after Fiji was placed under our Colonial Government, the chief Thacombau arrived at Levuka in H.M.S. *Dido*, on his return from Sydney. While there he or some of his party had measles. On January 6, during the voyage home, one of his sons and a native attendant fell ill with measles. They were treated in a house built for them on the ship's deck, and made so good a recovery that no obstacle was raised to their landing on the 12th. Two or three days after landing, another son of the chief returning with him was seized with measles. At this time visitors from all parts thronged the house where he was sick. On January 24 and 25 there was a great assemblage of native chiefs, some from the more distant parts of the large island. A strong force of native constabulary attended. Any new cases of measles in the native village had not at this time attracted notice. On February 12 a despatch from Mr. Layard announces measles to be epidemic among the natives, and that nearly one hundred of the native constables were down with it. It is evident that no alarm had been raised by February 7, or return labourers would not have then been despatched from Levuka to Malicoto, carrying the disease with them, as happened. On February 25 quarantine regulations were enforced, as it is said, "to prevent the spread of the disease to other islands; but to stay its progress here is impossible". The death of the most influential chief who attended the great gathering is reported at the same time. In the interior of the great island Mr. Harding, who stayed there at the risk of his life, says, "All the chiefs who attended the great meeting have it, and it is spreading rapidly." On March 3 Mr. Layard writes: "The attacks have been so sudden and complete that every soul in the village will be down with it at once, and no one able to procure food, or, if procured, to cook it for themselves or others. The people have died of starvation and exhaustion in the midst of plenty." On March 8 a missionary writes from Navulod

that measles has carried off a great number of natives, principally adults. The visitation has been more like that of cholera or plague than that of measles.

In June the Colonial Secretary reports the disease to have been carried everywhere by the latter end of March or the beginning of April; from that time it gradually decreased, and by the end of May it seemed to have died out. In three districts a census of the population had been taken before the epidemic; the loss since the introduction of measles was now ascertained. In Overlau: population 1,546 before, 1,099 after—deaths 447. Koro: 2,543 before, 1,855 after—deaths 688. Bau: 7,925 before, 5,711 after—deaths 2,214. From other islands 1,637 deaths had been returned from measles. The estimated loss is of from one fourth to one fifth of the native population, or more than 20,000 deaths in four months from this disease. The favourable progress of the early native cases negatives the idea of special proclivity. So, during the worst of the epidemic, at places where the people seized with fear had abandoned their sick, and some had died in such close huts that the walls had to be destroyed to let the trade-wind purify the air, the fact of only one death occurring among a number of cases treated in separate rooms with fair attention, is against any but a locally acquired intensity of the disease. Fear added to its effect, for in one place 26 deaths are said to have occurred within 48 hours of the seizure. The weather was unfavourable, for though the mean temperature was 80 deg., with a daily range of only 15 deg., except on the high grounds, tropical rains followed the hurricanes, and in March it rained during 204 hours, 50 inches falling in 27 days. The people choose swampy sites for their dwellings, and whether they kept close shut in huts without ventilation, or rushed into the water during the height of the illness, the consequences were equally fatal. Excessive mortality resulted from terror at the mysterious seizure, and the want of commonest aids during illness; there were none to offer drink during the fever, nor food on its subsidence. Thousands were carried off for want of nourishment and care as well as by dysentery and congestion of the lungs; the worst dangers from overcrowding were incurred in the small houses, and the worst dangers from cold by the sufferers rushing to the water, where they would continue immersed. Measles in these islands, in 20 deg. S. Lat., or within the tropics, corresponds very closely to what is recorded of its conveyance to the Faroe Islands in 70 deg. N. Lat., or near the Polar circle. The epidemic only ceased when every person had been

attacked. All the innate dangers of the disease here revealed differ only in degree and not in kind from those which are so commonly guarded against among ourselves by simple nursing. We need invoke no special susceptibility of race, or peculiarity of constitution to explain the great mortality. To people closely related to ourselves a similar fatality from measles has occurred. During the late Civil War in the United States, the numbers attacked with measles among the new levies exceeded 38,000, and the mortality attributable to this disease in two large hospitals was 20 per cent. This rate of mortality was doubled among the Garde Mobile in the month of January 1871, during the siege of Paris, when of 215 cases of measles 86 died, or exactly 40 per cent., the proportional mortality from measles exceeding that from small-pox.

ON INFANTILE DIARRHŒA.

By JOHN McKENNA, M.D.

(*Read: January 10th*, 1877.)

I WISH to bring before the notice of this influential society, and through it of the profession at large, a motive power in the production of a certain class of diseases and a natural means for their relief.

The diseases alluded to are congestive fever, dysentery, cholera, and diarrhœa. The influence concerned in producing them is heat, the antidote is cold, a natural specific as applicable to the prevention of infantile diarrhœa as vaccinia of small-pox. The best illustrations are drawn from diarrhœa as it occurs in children.

Twenty years' residence in equally hot but otherwise salubrious parts of Australia and of South America convinced me that the climatic change alone determined the æstival diarrhœa of children. Both in Buenos Ayres, lat. 34 deg. south, population 100,000, where I resided thirteen years, and in Melbourne, lat. 37 deg. south, where I lived for eight years, the concurrence of diarrhœa with hot seasons is as noticeable as it is in London, and is more strongly marked. In Buenos Ayres there was no registration of deaths during the time of my residence, but the appearance, nature and duration of diseases were the same as in Melbourne, Victoria, except that cerebral affections in infants were more frequent. In both these countries, it may be said that disease was almost unknown for nine months of the year in this class of subjects. From the late autumn, onwards through the winter and the spring, the climate is indescribably beautiful, except during the occasional winter rains. About the middle of the first summer month, December, when the weather begins to get hot and the warm days contrast with the cold nights, diarrhœa first appears, becomes worse in January and February, worst in the first weeks, and begins to decline, and ceases about the middle of March.

Some four years ago, after my arrival in Melbourne, the

Registrar-General of the colony published in the daily journals a statement that the deaths of children at and under three years equalled the births, and he called on the medical profession to propose means of stopping this calamity, and it was also then shown that the mortality almost entirely arose from diarrhœa and dysentery in the three summer months, viz., of December, January, and February. I immediately applied to the office in Melbourne and to the Registrar-General of all the colonies, and with the shortest delay was furnished with the information embodied in the following tables, which are taken from a paper on the Mortality of Childhood in Victoria, read before the Royal Medical Society of Victoria, and published in their *Transactions* for April 1878:—

Table I.

Showing the causes of death of children under three years of age in Melbourne during the three winter and during the three summer months, 1857:—

	Winter months. June, July, August.	Summer months. December, January, February.
Cerebral Affections	7	13
Convulsions	5	16
Debility	17	21
Marasmus	4	8
Bronchitis and Pneumonia	9	5
Teething	1	24
Diarrhœa	1	59 (1 English Cholera.)
Dysentery	1	

Some of the cases returned as teething suffered from diarrhœa.

Table II.

		Winter, 1857.	Summer, 1857-58.
Adelaide	Teething	0	33
South Australia	Diarrhœa and Dysentery	15	78
Population 33,000.			
Melbourne	Teething	4	27
Victoria	Diarrhœa and Dysentery	15	302
Population 100,000.			
Sydney	Teething	9	64
New South Wales	Diarrhœa and Dysentery	1	35
Population 53,000.			
Hobart, Launceston	Teething	1	9
Tasmania	Diarrhœa and Dysentery	4	52
Population 36,000.			
		49	600

The second table shows a proportion of more than twelve deaths from diarrhœa in the summer months to one death in the winter months.

Having discovered these surprising facts, remembering also the pathological characteristics of summer diarrhœa, and being satisfied that there was no agent which by direct contact could produce the simple congestion of which it consists, I was led to regard the stimulating effect of heat on the nerves and vessels of the skin and mucous membrane, exhausting the vaso-motor power, as an efficient cause. The action of the skin having been strongly excited by the increased heat of the day, fatigue and chill readily throw the blood in upon the relaxed vessels of the bowels, and so produce the congestion, named, in its mild form, diarrhœa, and in its more severe, dysentery.

That it was a simple congestion entirely unconnected with inflammation of any kind or degree, ample means were present to prove. In the exceeding dry and very hot summer, protrusion of the often highly congested bowel, but unaccompanied by any other symptom, was common in male adults, while in females of the better class it was often most troublesome.

In children it was so general and so persistent that from the middle of December till that of March the gut, when returned, could not be retained, except for a few minutes, in its place; and I have frequently seen spots of superficial ulceration on its dry and shining surface, though adding apparently nothing to the child's discomfort. Therefore I hold it to be incontrovertible that the æstival diarrhœa of children is a passive congestion of the mucous coat of the bowel, entirely free from any active irritation or inflammatory influence existing within itself.

This congestion is situated in the rectum, which is then supplied with the largest amount of blood vessels, and rapidly declines as it rises above the sigmoid flexure of the colon. The outline above given, however short and imperfect, meets the objects of this paper, except in one respect, viz., the omission of the conclusive proof that the quality or force assigned to the influence of the motive power of "climatic change", "atmospheric change", is of itself sufficient to create the diseases in question.

This, then, is the simple but true and exact description of the nature of æstival infantile diarrhœa, and will, I hope, banish from our literature that firstly, bad smells and putrid emanations; secondly, that raw fruit and undigested vegetable matter; and, thirdly, that teething is the omnipotent and sole cause of infantile diarrhœa.

The proof lies in the specific remedy that I now wish to commend to your notice as the cure of this summer disease; this is the use of cold, the action of which on the child is instant, firstly, in stopping the vomiting often present in extreme; secondly, in banishing thirst; and, thirdly, in cutting short the alarming wakefulness, and immediately procuring a sleep of many hours' duration; and, lastly, of gradually diminishing the dejecta, so that within a week the infant is restored to health.

REMARKS ON THE CHOLERA EPIDEMIC OF 1875 IN INDIA.

By ROBERT LAWSON, Esq.,
Inspector - General of Hospitals.

(*Read: March 14th*, 1877.)

THE Society was recently indebted to the Sanitary Commissioner with the Government of India for a copy of his Report on the Cholera Epidemic of 1875. This epidemic was both wide-spread and severe, and following as it did a period of remarkable exemption, for two years, of the greater part of Hindostan, the facts given in the Report are of extreme value in displaying the different steps by which the disease reinvaded the districts previously exempted, and a notice of their more prominent features will prove both interesting and instructive.

Recent writers on cholera in India regard the alluvial district through which the Ganges and Brahmapootra run in the last part of their course, as the locality from which the various epidemics proceed. This district, in which the disease seems perennial, and at some part or other of which it may be found every month of the year, has been denominated the endemic area of cholera; here it undergoes marked exacerbations in frequency, becoming epidemic about every fourth year, and then it generally extends along the vallies of the Ganges and Brahmapootra, spreading into the provinces of India to a variable extent in different epidemics, and at times passes beyond the limits of Hindostan and invades other countries. So far the different writers seem to agree; none of them venture to explain why the disease becomes epidemic in the endemic area at regular intervals, and they differ widely as to the mode in which they believe it to be transmitted to other localities on these occasions.

Dr. Bryden, proceeding on the basis enunciated by Jameson in his Report on the epidemic of 1817-19, considers that cholera is due to a material miasm, which is produced in larger quantity in the endemic area about every fourth

year, from which it is carried by moist winds to other localities, where it induces the disease, and, when it meets with a suitable soil, a portion of the miasm forming the seed for a new growth, a second crop arises, which keeps up the disease at the place and affords a fresh supply of the miasm which may be transmitted onwards to others, as yet free from it, where, should the local and meteorological conditions be suitable, a similar process is repeated; when these are not favourable, however, a miasm deposited at one season may remain dormant until the advent of another which is characterised by the necessary moisture, when the miasm, like other vegetable organisms, resumes its activity, and runs through the course just described. In this way he considers the cholera is carried from the endemic area to the north-west by the south-east winds from the Bay of Bengal during the period of the south-west monsoon, and from the same to the westward, through Central India and the Central Provinces, to the West Coast at Bombay, and southwards through the Deccan to Madras, and from that to Ceylon, by the easterly and northerly winds which prevail during the north-east monsoon, and more or less, and to a variable extent, at other times. When cholera has reached the west coast, the moist south-west winds may equally carry the miasm from the districts in the vicinity of the lower Nerbudda to the north, and north-east, to those portions of Central India which are not within the range of the easterly winds, passing over localities where the miasm is ready for diffusion. Dr. Bryden says, in the Bengal Presidency the miasm is never distributed by a dry atmosphere, and adduces evidence to show that the hot winds which prevail at certain seasons repress the evolution of cholera in the localities where they are experienced. Jameson (as quoted by Bryden), alluding to the connection between the easterly winds and the dissemination of the pestilential virus, wrote—"Of the nature of that connection we cannot speak certainly, nor can we tell whether these currents, acting as a vehicle of the poisonous matter, carried it along with them from one infected spot to another previously healthy, or acted merely from their superior moisture in the light of a strong exciting cause, eliciting the disorder in places where the virus had previously existed, although it were not yet brought into action."

Mr. Cornish, while he admits that the cholera epidemics of India generally originate in, and proceed from the endemic area, pursuing the courses indicated above, dissents altogether from Dr. Bryden's views as to the mode in which

they are disseminated. Instead of arising from a miasm, carried by the wind, he maintains they not unfrequently advance directly against the wind, extending marginally from a district already affected into others in the vicinity, the active agent in carrying the virus being man himself, while moving through the country on his ordinary avocations. Mr. Cornish admits that there is a form of cholera met with sporadically in Southern India, undistinguishable from the epidemic disease save that it shows no disposition to spread; but I am not aware that he recognises any locality in the provinces under his superintendence as being the seat of endemic cholera, in the same sense as he regards the endemic area in Lower Bengal. The returns of deaths from cholera for the last ten years (1866 to 1875), however, show these in the district of South Arcot every month, except in two instances, and in that of Tanjore every month, except in six instances. Some of these deaths may have been reported erroneously as the result of cholera, but the mortality from this cause was generally so considerable in these districts, even when at a minimum elsewhere, that there seems much reason to conclude the disease is really endemic in them.

Dr. Bryden based his generalisations mainly on the sick returns of the European and native troops, and of the jails, in the Bengal Presidency, up to the period when the death returns for the civil population became available. The sickness among these classes being derived from small bodies of men, at numerous points scattered over the country, can be regarded, however, as he justly observes, as indices only of what was going on among the general population around them; and though they afford a sufficiently correct view of the ordinary course of the epidemics, they are less suited for illustrating the spread of the disease over the country than the monthly returns of deaths among the general population, which are now published. The immunity from cholera in 1874 over nearly all Hindostan, followed by its general diffusion in an intense form in 1875, present us with a favourable opportunity of examining, with the aid of these returns, how far the manner of invasion in the latter year coincided with either of the views noticed above, as well as with some others, which are still unsettled.

In 1874, cholera was severely epidemic in the endemic area and its vicinity, and the western border of the outbreak reached a line passing from Ganjam, on the coast, to Benares, and from that, a little east of Fyzabad, to the base of the Himalayas. On the east, the epidemic extended into Assam,

and there was a considerable number of deaths from the disease in Burmah, though less than one-eighth of those returned there in 1873. There were also a small number of sporadic cases in the Tanjore (33), South Arcot (19), and Malabar (13) districts in the Madras Presidency in 1874, but elsewhere, throughout Hindostan and in Ceylon, there may be said to have been complete immunity from the disease. In 1875 the picture was exactly reversed. Cholera was experienced to the north-west, as far as Lahore, and the whole Peninsula was covered with it, from latitude 24 deg. southward, except a portion of the Central Provinces and the Vizagapatam, Godavery, and Kistna districts on the east coast, and that of Kanara on the west coast. Ceylon also was under the epidemic, though not severely; while in Burmah, up to November inclusive, there were only 155 deaths from it, though in December alone these rose to 606. Arranging the different provinces from north to south, as they stand on the map, the deaths from cholera in the two years were:—

	1874.	1875.
In Punjaub	78	6,246
North West Provinces and Oudh ...	6,464	64,427
Bengal proper and Assam	73,354	116,606
Rajpootana, Hyderabad, and Central India	4	14,649
Central Provinces	14	14,643
Berar	2	22,465
Bombay	37	47,573
Madras and Mysore...	313	97,051
Ceylon	—	1,817
Burmah	960	761

If, now, we examine the steps by which this extensive invasion took place, the first indication is found in Ceylon, where, in January, 16 deaths were caused by cholera in the town of Colombo; in February there were 57 deaths there, 9 at Negombo, north of Colombo, and 1 at Galle, the packet station at the south-west point of the island; in March there were 194 deaths at Colombo, 48 at Galle, 25 at Negombo, and 14 at other points. During these months there were only 30 deaths registered from cholera in the whole of the Madras Presidency, of which 5 were in the Malabar district, on the west coast, 2 were in Ganjam, on the east coast, in the extreme north, and 23 in the districts of Tanjore, Trichinopoly, and South Arcot, also on the east coast, but to the north of, and not in immediate communication with, Ceylon. Of the latter, 7 occurred in January, 13 in February, and 3 in March.

In January, cholera was at Cuttack and Balasore, at the

south-west corner of the endemic area, and less active from that point, through the whole of the low alluvial tract to the north-east, while sporadic cases in small numbers were met with on the slopes all round the hilly tract of Chota Nagpore, as well as along the Ganges, as far as Benares and Allahabad, and to the north-west, up to Agra and Meerut. In February the disease became active in Benares, Jounpore, Azimgurh, Mirzapore, Allahabad, and in Pertabgurh in Oudh, which is contiguous to those districts, and four other adjacent districts in Oudh presented sporadic cases. In the south, Poree, adjacent to Cuttack, was affected in February, and 7 deaths were registered at Bilaspur, considerably in advance of the line reached by the epidemic in 1874. In March, the disease became intensified in the endemic area in the districts along the Ganges and in Oudh, where it had been the previous month, and became more active at Agra and Budaon, to the north-west, also at Bilaspur, where there were 77 deaths this month. It appeared at Raipur, still further to the west, and in Rewah, south of Allahabad. In the Nasick district of the Bombay Presidency, near the west coast, 10 deaths occurred in March.

In April the disease manifested great activity, not only was it intensified in most of the localities previously affected, but it broke out at Banda and Hamirpur, west of the Jumna, and appeared in a milder manner at Muttra, Bulandshahr, Meerut, and Bijnor, in the north. It continued at Bilaspur, but there was no death at Raipur this month; and at Nasick, in the Bombay Presidency, the deaths rose to 481 this month, while the epidemic appeared in the districts of Khandesh, Kaira, and Panch Mahals, also in the contiguous native states of Western Malwa, Indore, and Bhopawar, and in Hoshungabad, Nimar, and Burhanpur, all to the north of Nasick; in those of Ahmednagar and Poona, to the south of it; and in Bombay city, where, up to this time, there had been only a single death from cholera in January, and another in February. There were 8 deaths in the Ratnagiri district on the coast, some distance south of Bombay, in April. In Madras, in April, the deaths from cholera had increased to 65 in the Tanjore district, and there were 9 deaths in that of Tinnivelly, in the extreme south, and a few sporadic cases in South Arcot. In Ceylon the disease continued in Colombo, Negombo, and Galle, but in no great force, and in Burmah 25 deaths occurred at Amherst, where there had been a single fatal case in January.

In May the epidemic continued to increase in intensity where it had already appeared, and to extend to fresh points,

not previously occupied, or at which a few sporadic cases only had been seen. In this way it appeared at Burtpore, Delhi, and Rhotack, and there was a considerable outbreak, including Saharanpur and Dehra, with lesser indications, in the mountainous districts of Kumaon and Garhwal. This month the disease became very active at Bilaspur, and re-appeared in force at Raipur. In the Bombay Presidency, Ahmedabad, adjoining Kaira, was affected in May, and Broach, lying between the latter and Khandesh. Cholera continued in Western Malwa and Bhopawar, at Hoshungabad and Nimar, and a few cases appeared at Gunah, to the north of these places, and Betul, to the south, and at Akola and Buldana, just east of Khandesh. The district of Tanna, on the coast north of Bombay also was covered by the epidemic. In the Madras Presidency there were 1,589 deaths in the Tanjore district in May, 30 in that of Tinnevelly, and a few sporadic cases in South Arcot and Trichinopoly. Colombo, Negombo, and Galle, in Ceylon, continued affected, and Amherst, in Burmah.

In June, cholera continued active in most of the localities already indicated, and appeared at Meerut, Umballa, and Kangra, in the north, and to a slight extent at Gwalior. There were a few cases at Narsinghpur, near Jubbulpur, and at Bhandara, near Nagpur. In the Bombay Presidency cholera appeared at Surat, which lies between Khandesh (affected in April) and Broach (which commenced to suffer in May), Jaulna and Aurungabad, which adjoin Khandesh, and to the east of these Basim, Amraoti, and Ellichpur were also affected at this time. Colaba, on the coast south of Bombay, was affected in June, also Satara and Sholapur, to the east of the ghauts south of Poona. In June, too, Haidrabad, in the Deccan, came under the disease. In the Madras Presidency the deaths from cholera in the Tanjore district amounted to 4,972 in June, and it had become epidemic in the adjoining districts of Trichinopoly and South Arcot, and attacked Coimbatore to the west and Cuddapah to the north, and a few sporadic cases were met in Chingleput, the district in which Madras town lies. In Ceylon the disease continued at Colombo and Negombo, and appeared in the Damboola district, and at various places in the centre of the island, and in the district of Manar, in the north-west. In Burmah, Amherst continued affected, and sporadic cases occurred at Bassein, in the delta of the Irrawaddy, and at Akyab, on the coast to the north.

In July, in the north, cholera extended to Sirsa, Karnal, Ludhiana, Amritsar, and commenced at the mountain station

of Simla. It also appeared to a slight extent at Sambalpur, east of Bilaspur, and broke out at Nagpur and Wardha. In the Bombay Presidency the districts of Belgaum and Kaladgi, to the south, were first affected this month, and in the coast district of Ratnagiri, where there had been sporadic cases since April, the disease became a little more frequent. In the Madras Presidency the districts of Madura and Salem, on the south, were attacked. Chingleput and North Arcot presented sporadic cases. Nellore, the district east of Cuddapah, where the disease was now epidemic, became affected, and Bellary, west of Cuddapah, and between it and Kaladgi, showed the first indications of the disease. In July alone the deaths from cholera in the three districts of Tanjore, Trichinopoly, and South Arcot, were 12,369 in a population of 4,930,000, or 2·5 per thousand. In Ceylon the disease was less prevalent in the south, but it acquired some intensity in the Manar district, in the north-west.

In August, there were some further extensions of the disease in the north. Lahore and Gurdaspur were affected. In the central provinces, Saugor, Chindwarra, and Chanda were attacked, and adjoining the last Wun, a district of Berar. In Bombay cholera became epidemic in Ratnagiri, on the coast. In Madras there were a few sporadic cases in Ganjam, the northernmost district on the coast, contiguous to Pooree, in July and August. In the latter month Chingleput and Madras town came under the epidemic, also Kurnool, between Cuddapah and Haidrabad, in both of which the epidemic was now in great activity. Bellary and North Arcot, where in July there were only the first indications of the disease, were now fairly under the epidemic. Mysore also was affected in August, and the district of Malabar, on the west coast. In Ceylon the disease extended to several other places, though in no great force.

In September, there was some increase of the disease at Lahore, also in the Malabar district, where it went on to a severe epidemic in November and December. Dharwar, the southernmost district of the Bombay Presidency, to the east of the Ghauts, was first affected in September, and had a severe epidemic the following month. In Ceylon, in September, the disease appeared at a considerable number of places, apparently on the eastern side of the island, but was in no great force anywhere; and in Burmah, where the outbreak at Amherst had ceased in August, it appeared in November at Rangoon, and the following month at some other points, embracing Tavoy in the south, and Thoukwa

in the north, causing in these months 635 deaths out of a total of 76, returned for the whole year.

In the Bombay Presidency, the districts from Poona northwards mostly showed the first indication of the epidemic in April, and Poona, Nasick, Ahmedabad, Kaira, and Panch Mahals, had the greatest mortality in May and June, after which it declined. In Khandesh and Ahmednagar, which are more to the eastward, the mortality increased till July, after which it declined; and still more to the east, Aurungabad, and Berar, and Satara, and Sholapur to the south, had the most severe part of the epidemic in July and August, as also Raipur, east of Nagpur. The disease was at its height in August and September down the centre of the peninsula, when Nagpur, Wardha, Haidrabad, and, in the Madras Presidency, Cuddapah, Nellore, and North Arcot, suffered most. In Tanjore there was a great mortality in June, and the epidemic culminated in July, as it did in Trichinopoly also; in South Arcot, July and August were the worst months; in Madura to the south, and Coimbatore to the west, the deaths increased greatly in October, culminated in November, and fell considerably the following month. Tinnevelly, south of Madura, and Salem, north of Coimbatore, showed a prevalence partaking of both periods, a pretty high death-rate in September being continued without much variation till November, followed in Tinnevelly by a large increase in December; this month, also, the deaths in Tanjore, Trichinopoly, and South Arcot, were somewhat increased. In Malabar, on the West Coast, where the disease had appeared in August, it gradually increased until December, when 1,575 deaths were registered from it. Dharwar, the southernmost district of the Bombay Presidency, was first affected in September, and, in October, 1,056 deaths were registered, declining to 539 and 477 in November and December. Belgaum to the north of Dharwar, and Bellary in Madras, which adjoins it on the east, like most of the other districts down the centre of the peninsula, had a considerable number of deaths in August and September; in Bellary, the same rate continued into October, while, in Belgaum, the deaths in October were 1,460, more than thrice their number in September; the Kaladgi district, which also adjoins Dharwar, and lies between Belgaum and Bellary, merely presented the maximum of August and September, after which the disease abated considerably.

In the West Coast districts, Tanna, at the south of which Bombay city lies, had a considerable epidemic, which was

about equally severe from June till August. E
had a moderate outbreak, which was at its heig
In the Colaba district, south of Tanna, the dise
ated in August; Ratnagiri, south of Colaba, su
here the greatest mortality was in August and
North and South Kanava, which intervene betw
giri and Malabar, had only a few cases, the fc
October to December, the latter in November
the Bengal Presidency, the course of the epi
similar to that so fully described by Dr. Bryden
epidemics, so it is unnecessary to detail it here.

The important points brought out in this recit
at the beginning of 1875, when for more than twel
there had been very little trace of cholera an
Southern and Western India, and a complete fre
it as an epidemic to the west and south of a line
from Poore to Benares, and from thence a little t
ward of Fyzabad to the Himalayas.

1. Cholera appeared in Ceylon about the comn
of the year, and pursued a course leaving no dc
epidemicity, though in a mild degree.

2. That this disease became active west of th
area in February, and the first indication of its
to the southwest was met with at Bilaspur this m

3. While in March cholera had advanced south
Allahabad into the Rewah territory, and west
Bilaspur to Raipur, it commenced in the district
close to the west coast of the peninsula, without
of the disease in the intermediate country; the
Nasick were, in March, 10, in April, 481, and, in M
In April, cholera began in the districts of Khand
north of Nasick, in those of Ahmednagar and Po
south, and in Bombay city; also in Kaira, a
Mahals, two districts in Guzerat at the top of tl
Cambay, in Western Malwa, and Indore, to th
Panch Mahals, and still further east, and south of
budda, at Nimar (21 deaths), and Hoshung
deaths). From these points eastward to Rewah a
no death was recorded up to April inclusive, excep
one at Jubbulpore that month. There were thus
two epidemic centres in the west, neither having c
with the epidemic in the east, by a continuous de
of the disease through Central India, or the ce
vinces.

4. In the Madras Presidency, cholera commen
epidemic in the Tanjore district in April also, dc

sporadic cases having been reported here every month from the beginning of 1874, except February that year. In April it appeared, though slightly, at Tinnevelly, at the extreme south of the peninsula. Tanjore is separated from Poona and Raipur, the nearest points in the north, where the disease was in April, by about 600 and 700 miles respectively of country at this time without a trace of it.

5. Cholera appeared at Amherst in Burmah in April.

6. There were, then, besides the epidemic in progress in the Valley of the Ganges, four other points attacked, at great distances from each other, and from the endemic area, with which they were not connected, by a continuous manifestation of the disease. In Bombay, Madras, and Ceylon, the disease soon appeared at other points round those first attacked, though frequently at considerable distances from them; these ultimately coalesced, and the epidemics from the east and west portions of the Deccan, north of the Kistna, met in the centre from Nagpur to Haidrabad, and culminated in August and September; while that in the south, which now covered nearly the whole width of the peninsula, reached the Kistna at the same time.

These facts place it beyond doubt that cholera may arise and become epidemic in Ceylon without being conveyed from the continent of India in its vicinity; that it may also spring up in parts of the Madras Presidency without any trace of its being conveyed from the endemic area in Lower Bengal, through the central provinces and the Deccan, for at least two years previously (for there was no indication of such progress in 1873); and, that, even in the Bombay Presidency, the outbreak of 1875 had no evident continuity with that occurring at the same time in the Valley of the Ganges. The importance of these positions on our explanation of the course of cholera epidemics in India is obvious; without excluding the possibility of the transference of a miasm by successive stages from the endemic area, by winds or otherwise, on other occasions, they bring out clearly that virulent epidemics may spring up at various points in that country without any continuous connection with other places where the disease may be prevailing; it, therefore, becomes necessary for us to reconsider the modes of extension of epidemics in India hitherto recorded.

It was mentioned above, that none of the writers on cholera in India had ventured to account for its regular exacerbation about every fourth year in the endemic area, neither have they taken the causes of this exacerbation into account in their explanations of the mode in which it is dif-

fused over the country; though a moment's consideration will make it clear that the factors which lead to the epidemic development of the disease in the endemic area must be intimately connected with its epidemic activity elsewhere as well, and every theory on the subject, which does not take cognisance of the operation of such factors, must be defective and misleading. Influences similar to those which determine the increase of cholera in the endemic area, and its activity along the Valley of the Ganges, and up to the northwest in 1875, evidently acted through the whole length of the peninsula to Cape Comorin, and in Ceylon, and led to the violent epidemic of that year, over a country that, during the greater part of 1873 and 1874, was practically free from cholera.

It may be supposed that the extensive system of railways, now in operation in India, contributed materially to the diffusion of cholera in 1875; but the Sanitary Commissioner is opposed to this view, and the facts afford it no support. Dr. Bryden has shown that, in the Bengal Presidency, the areas occupied from year to year in the successive epidemics, have remained unchanged notwithstanding the introduction of railways, and an epidemic starting from the endemic area now takes quite as long to reach Delhi or Lahore as it did fifty years ago.

The occurrences of 1874 illustrate this point. That year a severe epidemic advanced as far as a line running from Poona to Benares, and from thence northward, a little to the eastward of Fyzabad, to the foot of the Himalayas; the railway from Calcutta to Allahabad crossed this line at a right angle, and was powerless to extend the area of the epidemic towards Allahabad, far less to carry it to the north towards Agra or Delhi on the one hand, or to the south west towards Bombay on the other. A branch of the Oudh railway runs from Benares to Fyzabad, skirting the edge of the cholera field there, but without carrying the epidemic to Fyzabad, or extending it along its course to the northwest of that place. In 1875, also, though cholera was at Allahabad in February, and it appeared in the Nasick district in the Bombay Presidency in March, at this time no intermediate point on the railway between these was affected; in April, Jubbulpur, an important station on the line, had a single death, and in May a second, and in September and October nine others, in all eleven during the year. Narsinghpur, another important station, was first affected in June, and had only ninety-nine cases during the year; while Hoshungabad and Nimar, farther west in the

valley of the Nerbudda, and noticed by Bryden for their liability to cholera at an early period of the year, long before the railway was opened, were affected in April. In the west, again, though cholera was in Bombay in April, and in Kaira, and Panch Mahals, the same month, and the Baroda railway, which connects these districts, runs through those of Broach and Surat, lying between them, yet cholera did not commence in Broach until May, nor in Surat before June. Proceeding southward, though cholera was at Poona in April, it did not reach Sholapur, the next district to the south along the railway, before June, in which month, also, it commenced in the Satara district, west of Sholapur, but away from the railway; and at Haidrabad to the east, which is in railway communication with Sholapur and Poona. In Madras similar occurrences are met with.

Another feature in the distribution of cholera, particularly noticed by the Sanitary Commissioner, is brought out very distinctly by returns of the registration circles in each district, and of the number of villages in these circles, to which is added the number of circles invaded by the epidemic, and of the villages in each that suffered; a form of return commenced some years ago by Mr. Cornish in Madras. The tables are too extensive to be introduced here, but their nature will be shown, and their value indicated by the following abstract of those for Oudh (exclusive of the district of Lucknow, for which the figures are incomplete) and Berar, the former of which lost 22,070 from cholera out of a population of 10,396,590, and, the latter, 22,465, out of a population of 2,184,945, or, 2·11 per 1,000 living in Oudh, and 10·28 per 1,000 in Berar.

	Total No. of Registration Circles.	No. of Circles attacked by Cholera	Total No. of Villages.	No. of Villages attacked by Cholera.
In Oudh... ..	125	... 120	... 23,738	... 4,042
Per cent. attacked		... 96	...	... 17
In Berar... ...	131	... 98	... 5,427	... 2,431
Per cent. attacked		... 75	...	... 45

Thus, while 96 per cent. of the registration circles came under the epidemic in Oudh, showing its causes to have been active over nearly the whole country, there were only 17 per cent. of the villages attacked; on the other hand, in Berar, while only 75 per cent. of the circles manifested the epidemic, 45 per cent. of the villages were attacked, and the mortality, as already stated, was nearly five times greater. The other returns for the Bengal Presidency in 1875 bring out the corresponding features everywhere, and

their existence in the Madras Presidency was pointed out by Mr. Cornish as long ago as 1872.

These facts show conclusively that, even where the epidemic was most intense, the combination of conditions necessary to excite the disease was often confined to very circumscribed localities, and that points of immunity were mixed up with those which suffered all over the country to a varying extent in different districts.

As the social condition of the people in both the infected and exempted villages did not differ, while free communication went on among them, the Sanitary Commissioner considers the facts just referred to irreconcilable with the theories which ascribe the chief part of the diffusion of cholera epidemics to the agency of man himself, a position deserving of our most careful examination.

ON THE INTRODUCTION OF ASIATIC CHOLERA INTO THE UNITED STATES IN 1873.

By ELY McCLELLAN, M.D.,
Surgeon United States Army.

(*Read: April 11th*, 1877.)

Early in the Spring of 1873 it was announced that cases of Asiatic Cholera had occurred in the city of New Orleans, Louisiana, the first recognised case being on the 9th of February. By the first of July of the same year the disease had been diffused over the valley of the Mississippi river, and the states of *Louisiana, Mississippi, Arkansas, Tennessee, Illinois, Missouri, Kentucky, Ohio, Indiana, Alabama, West Virginia, Georgia,* and *Minnesota,* had been subjected to the influences of the epidemic. Upon them the severity of the outbreak was expended; to them the epidemic influences seemed to be almost entirely confined; for although during the succeeding two months the states of *Pennsylvania, Texas, Iowa, New York,* and the territories of *Utah* and *Dakota* were invaded by the disease, the demonstration was by no means so severe as in the states first indicated.

By some individuals this demonstration of the disease was looked upon as an outbreak of pernicious or congestive malarial fever, by others it was recognised as endemic cholera; but by the vast majority of the medical men who encountered the disease it was classed as Asiatic cholera, and they contended that the epidemic differed in no way from other demonstrations of the disease in this country.

After a careful survey of the entire area of infection, and the study of the record of 7,356 undoubted cases of the disease, we had no hesitation in pronouncing the epidemic to have been one of malignant epidemic cholera. In the study of this epidemic our judgment was undoubtedly influenced by certain epidemic explosions which it was our good fortune to witness during the period of 1873, in which the disease was diffused over the state of Kentucky. As

the facts exhibited by each of these outbreaks were so characteristic of the disease, and so strongly corroborative of events narrated of former epidemics, we would again relate them before proceeding to the subject of the introduction of the disease into the United States in 1873.

On the 10th day of August 1873, a man named Bewley, whose home was at Russellville, Tennessee, arrived at the town of Lancaster, Garrard County, in the interior of the state of Kentucky. Russellville at the time of Bewley's departure was infected with epidemic cholera, and this was the cause of his leaving his home—as we have been informed by Dr. C. D. Riggs of that town, who adds the further information that Bewley, prior to his departure, had suffered from a "suspicious diarrhœa". The journey was performed upon horseback, and although the man was sick several times upon the road, it was accomplished. On arriving at Lancaster, however, he was obliged to go at once to bed. Vomiting, purging, and cramps set in, followed by complete collapse, but after many hours he reacted and passed into the "tepid stage".

On August 14th, a negro man who waited upon Bewley was taken with cholera, and died after a few hours' illness.

On August 15th, the father-in-law of Bewley, who resided near Lancaster, died of cholera after an illness of eight hours. This man had visited Bewley, remained with him one day, slept with him one night, then, being taken ill, started for his home, but died before he reached it.

On August 16th, a negro woman living near the house in which Bewley was sick died of the disease, and it spread to negro cabins adjoining her house.

From these cases the disease spread and infected the entire town. A public well was so located in the rear of the house in which Bewley was sick, that it was a matter of almost impossibility that the water therein contained should not become contaminated. All who drank of this water suffered more or less severely from the disease. Bewley lived until August 22nd, but before his death nineteen fatal cases of the disease had occurred at the town.

The epidemic influences continued at Lancaster until the 5th of the next month (September), at which time the disease disappeared, save in a single instance.

On September 19th, fourteen days after the occurrence of the last case in the town, an aged lady named Guthrie visited Lancaster from her home in the country, at which she had remained completely isolated during the epidemic. She went to the house at which Bewley had been sick, was

given the room and bed in and on which he had died. The day after her arrival (Sept. 20th) she was taken with cholera, and died after an illness of thirty-six hours.

This narrative is considered valuable from the fact that the individual who introduced the disease into that community was the *twentieth death* in the epidemic, and it is therefore evident that had the investigation started with the first fatal case, the clue would have been lost. It also demonstrates the fact that the specific poison of cholera may become located in the meshes of fabrics, from which the contagion may at any time be given off, for in this epidemic outbreak the first and the last cases occurred in the same room and upon the same bed. From this room the first fatal case in the epidemic went to his home to die—from this room the second fatal case went to his home to die—in this room the twentieth death occurred—and in the same room the thirty-fifth death closes the record of the epidemic.

The second demonstration to which we would ask attention occurred in the town of Lebanon, and county of Marion, of the same state.

In a paper which we read before the American Public Health Association in 1873, we stated that the state of Kentucky had received the cholera infection that year by three distinct lines of advance.

1. By navigation on the Ohio river.
2. By railroad travel.
3. By a solitary traveller over a mountain highway. Of the third of these lines the instance has already been given, and the narrative which follows is an illustration of the second.

On or about the 14th of July 1873, a negro man, working upon the line of the Ohio and Cumberland Railroad (then in course of construction), at a point some six miles from Lebanon, was taken with acute but painless diarrhœa. He had recently arrived from the line of the Memphis and Paducah Railroad, upon which, at the time of his departure, cholera was epidemic. He was sick in one of a cluster of cabins upon a hill side. Immediately in front of and a few feet below these cabins was a spring, from which the occupants obtained water. On the 14th, 15th, and 16th days of July there was considerable rainfall, and by surface washings the basin of this spring was nearly filled with *débris*.

On the 17th of July two violent cases of epidemic cholera occurred in the cabin next to the one in which the man with the diarrhœa had been sick, and many others followed

rapidly. One of these cases, on the 17th of July, was a negro, whose home was in Lebanon, from which town his mother went to attend him during his illness, and when the patient was convalescent, he and his mother returned to Lebanon.

On the 19th of July a negro workman on the Ohio and Cumberland Railroad returned to his home at Lebanon and died of cholera.

On the 11th of August, the negress (noted as the mother of the case of July 17th) died at her home at Lebanon of cholera, after ten hours' illness.

On the 18th of August, a white man who resided in the vicinity of the houses in which the first two cases died, was taken with cholera, became profoundly collapsed, but finally recovered.

On the 19th of August, a young white woman living in the same vicinity had an attack of the disease, but recovered.

On the same day, a white man, who frequented this part of the town, died of cholera, after a few hours' illness.

On the 25th of August, a negro who had been among the workmen on the Ohio and Cumberland railroad, died of cholera in the same part of the town.

The houses at which these cases, save one, occurred were located in the lowest portion of the town. They were built along the line of a small stream, forming one of the natural drainage channels of the town. The house in which a case died on August 25th being built directly over the stream. These facts are of great importance in the investigation, as will be shown hereafter, and they are also of value, in consideration of the fact that although from July 18th to August 25th seven cases of cholera, five of which were fatal, occurred in the town of Lebanon, an epidemic was not instituted.

In these cases, with but two exceptions, no efforts were made to stamp out the disease. The medical men of the town, impressed with the false teachings which have been so fashionable in America, considered the disease to be of local origin, and could not be convinced that it possessed any infectious or contagious powers. Therefore disinfectants were not used. Two of these cases, however, those of August 18th and the man taken sick August 19th, being under my individual control, were subjected to careful disinfection.

On the 26th day of August the Marion County Fair was commenced upon the fair grounds near the town of Lebanon, and it was attended by many visitors from the adjacent

counties of the state. The water supply of these fair grounds being limited, a contract had been made to carry water from the town to supply the deficiency. A well was selected by the contractor in the western portion of the town, almost immediately upon the bank of the small stream which has already been noticed, and in the same block in which all the cases of cholera reported had occurred.

During the evening of August 27th a violent rain storm deluged the country; the small stream was filled to overflowing, and the well in question was flushed. The excreta of all of these cases of cholera had been thrown (without any attempt at disinfection, save in the two instances noted) upon the banks of this stream, and nothing but a direct interposition of Providence could have prevented the surface washings from contaminating the water contained in the well.

During August 28th and 29th the attendance upon the fair was large. The water from this well was served, as upon former days. No case of violent illness had occurred in the town since the 25th. A fatal security seemed to possess all. By those to whom we had communicated our apprehensions we were looked upon as an alarmist.

A public ball closed the festivities, and the inhabitants of the town went to their rest as usual; but before daylight of August 30th the explosion occurred. Almost simultaneously thirteen fatal cases occurred, and wherever in the county of Marion, or in the counties of Washington, Nelson, Taylor, Adair, Boyle, Clinton, and Mercer, individuals resided who had attended this fair, the fatal disease occurred.

The disease in this demonstration occurred as frequently in isolated positions as it did in those thickly settled, the only prerequisite for its occurrence being attendance upon the Marion County Fair and the drinking of this water. Large numbers of persons visited this fair and escaped the disease, but they were those who drank no water upon the grounds.

It is not often in America that epidemics distinctly traceable to drinking-water occur, and these instances are advanced as marked illustrations of the agency of drinking-water in the diffusion of disease, as well as illustrations of the fact that in the diffusion of 1873 the disease obeyed the same laws as those known to have governed other epidemics.

These instances are recorded upon no hearsay evidence, as both of the towns of Lancaster and Lebanon were garrisoned by United States troops. In the discharge of our official

duties we were compelled to visit Lancaster, and Lebanon being our station, we were cognisant at the time of the events narrated. Many other equally well attested instances will be found in the report upon the epidemic of cholera in the United States during the year 1873.

At the meeting of the American Public Health Association, November 11th to the 14th, 1873, the distinguished President of the Board of Health of the State of Louisiana, presented a report upon the cholera epidemic as it affected the city of New Orleans, Louisiana, which recited the occurrence of the first fatal cases of the disease, described the sanitary condition of the city, and contained the statement that two cases, one of which occurred April 14th, and the other April 16th, *were the only two cases of cholera that occurred among or near to the shipping, or that had any connection with it.* [The italics are our own.]

Later, when the report of the State Board of Health of Louisiana was published, the subject was more elaborately treated.

The first fatal case occurred February 9th, the last, November 21st. Only seven cases occurred in the last six months of the year. The total number of cholera deaths in the first six months of 1873 was 244. The so-called multiple cases, that is, those cases occurring in the same domicile, were comparatively few in number, being but twenty in all, occurring in nine houses. The mortality during the prevalence of the cholera was at the rate of ten blacks to thirteen whites. No importation could be proven. No evidence of portability or of infection was exhibited. No general diffusion of the disease occurred among the population of the city, which amounted to about 200,000 souls. The disease appeared at the season of the year when intestinal disorders occur in that city. *Large numbers of persons being taken ill with profuse vomiting and purging, sometimes preceded by faintings and cramps, with great subsequent weakness, recovered without any aid whatever, save the vis medicatrix naturæ.*

These were the points upon which the dogma that the disease had a local origin was based; and to this opinion the vast majority of the medical men of that city firmly hold.

So wedded are a large number of American medical men, especially of the south and south-west, to the theory that epidemic cholera and yellow fever are diseases of local origin, resulting from certain telluric and meteorologic

influences, a falsity that has been perpetuated by certain prominent medical teachers since the acrimonious controversy which took place between the College of Physicians and the Academy of Medicine of Philadelphia over the yellow fever epidemics of 1793. Some honestly seek to prove this theory, but they are willing to accept no evidence as proven unless it in all respects coincides with their own views. To prove the infectiousness of cholera or yellow fever, it must be shown how every individual instance of the disease was infected; if this cannot be done, there is no infection. The first fatal case that occurred in a community must be the starting-point of the investigation; they view with suspicion and distrust all prior non-fatal cases, and combine to reject them. They demand that infection on ships arriving at healthy ports shall be *visible, tangible*, otherwise they deny its existence, although forced to admit by official records that such vessels had arrived from infected ports. Others will accept nothing, admit nothing, and defend their position by mere assertions.

While all admit the infectiousness of small-pox, scarlet fever, etc., and the impossibility of tracing or accounting for outbreaks of such diseases, they will not apply the same logic to either cholera or yellow fever. Circumstantial evidence (upon which the law takes lives) is not strong enough to account for the appearance of these diseases in America. Nothing will convince them, nothing satisfy their obstinacy.

Upon reading carefully the report of Dr. White, and critically examining his illustrative map, we were struck with two points which it seemed to us he had failed to grasp.

1. That the first twenty-five cases of cholera which were reported in detail all had a common point of infection, from which they carried the disease to various portions of the city.

2. That it was incorrectly stated (even accepting the points at which death occurred as starting points of the disease) that the cases occurred "*at many localities remote from each other*".

The report of Dr. White in almost every instance locates the point at which the subject of cholera worked prior to his sickness, which was upon the steamboat and ship levees. It was found that the point upon the levee assigned to steamboats was between that assigned to ships on one side, and ships and steamers upon the other. [The distinction made being between steam-vessels navigating rivers and *those built* for traffic upon the high seas.]

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Again, the map presented by Dr. White exhibits the point at which each of these twenty-five cases was treated for the disease. Now, by drawing upon the map a line from each of those points to the point at which he tells us they were employed when taken ill with cholera, it will be seen that the lines come together upon the steamboat and ship levees.

If upon this map a circle be described, the centre of which rests at the river foot of Canal Street, the great central street of the city, and the diameter be made great enough to include the case recorded at greatest distance from the river, it will include all of these cases. The heart of the city only will be included, the larger portion being without its limits. It is positively asserted that each of these cases had personal contact with ships and shipping, excepting those now noted.

Case iv was a female, who died March 1st of cholera. Her husband, an employée upon a steamboat, having died the previous day in the same house, of a disease which all the attendants describe as similar to that of the wife, although the attending physician did not call it cholera, but *acute gastro-enteritis*.

Case vi was not connected with the shipping, but he kept an eating-house (of low grade) upon the steamboat levee.

Case ix [in reality the record of *three* cases] occurred at no great distance from the levee. The man was a dissipated tailor, of whose wanderings no trace could be kept. The family lived in low damp premises, with stagnant and filthy water in yard. The man was taken sick, but recovered; the next day, the wife, who died; then their child, who recovered.

Case xiv. No connection with shipping was required, as an adult who was so connected died in the same house in a typhoid state after a sudden and violent illness.

Case xv. The father of this child was constantly called by his business to the steamboat levee.

Cases xix and xx were children who lived but one block from the lower ship levee.

It was, therefore, evident, that the statement that the cases of April 14th and 16th were the only two cases of cholera that occurred among or near to the shipping, or that had any connection with it, was, to say the least, inaccurate, for a positive connection is established in the great majority of these cases; while, of those remaining, each had been in contact with persons who were taken with the disease upon

the levee, save in the case of "the drunken tailor", whose wanderings were not to be traced. It naturally occurred to us that if such inaccuracies marked the report under consideration upon such an essential particular; if the record of the first twenty-five cases was indeed a record of thirty cases of the disease, five of which are untraced, would it not be possible to obtain an account of other cases, probably non-fatal, which might have occurred at an earlier date?

To this point our energies were directed, but without avail. We failed utterly to establish the arrival of any individual personally infected with cholera. We failed to find any other early cases than those already recorded. And upon this latter point we desire distinctly to be understood. We do not express an opinion that such cases did not exist—we are still of opinion that such cases did occur —that there were numerous non-fatal cases, but our efforts were fruitless in discovering them. Be it remembered that we were working against the prejudices of education in the majority of local professional men, and, while we acknowledge unbounded personal courtesy extended to us on all sides, we failed to induce any enthusiasm as to the investigation. The story had been written to their satisfaction, and that ended the matter.

But while making this acknowledgment of failure, our investigation established certain facts so significant, of such circumstantial weight, that the epidemic of 1873 is robbed for ever of its charm of originality, and must take its place amongst those in the oft-told story of epidemic diffusions.

Our attention was directed to the quarantine of the port of New Orleans. It was stated in the Report of the State Board of Health, upon the authority of the resident physician at the Mississippi River Quarantine Station, that, from January to July 1873, "there had passed and been personally examined 638 vessels of all kinds, and in no instance had there been cholera in any form".

Being desirous of obtaining more explicit information upon the character of this quarantine, we addressed to the officer in charge certain questions, and received in answer the following. "*Quarantine in the months you refer to was confined to asking each captain the enclosed questions.*" This list of questions were of the most general character; and the only other information obtained was, that from ports held in 1873 as cholera-infected, there arrived at New Orleans 175 vessels, conveying 3,500 men as crew, and 1,844

passengers, a total of 5,344 individuals arriving during the months of January, February, and March 1873.

Through the kindness of the President of the State Board of Health we were able to abstract the following information from the monthly reports of the Mississippi Quarantine Station—viz., from January to July 1873, there arrived at the port of New Orleans 769 vessels, with 5,163 passengers and 14,731 crew, making a grand total of 19,894 individuals. All the information we could obtain as to the Mississippi river quarantine confirmed us in our opinion that, during 1872 and 1873, it was utterly worthless and unreliable, that none of the statements made by the *then* quarantine officer should be received as undoubted facts. This officer was subsequently discharged for disobedience and negligence of orders, and for having passed a yellow fever ship up to the city without detention for disinfection. (See *Report of the Board of Health of Louisiana*, 1874, p. 13.) The quarantine station is so far below the city, that it is out of the direct control of the Board of Health, and they could not be held responsible for the performance of duty of an office to which they neither *appoint*, nor from which can they *remove*, the incumbent.

If, in 1874, this officer would wilfully endanger the health of the city with a yellow fever infected vessel, why should we be compelled to accept his statement as to 1873, and the inference therefrom, that he had passed no cholera infected vessel?

If he did not take the pains to discover the probable infection of yellow fever in 1874, would he naturally have taken greater pains to discover cholera infection in 1873? The facts brand for ever the Mississippi river quarantine of the winter of 1872-3 as having been worse than useless.

Failing to obtain reliable data from the sources which had as yet been investigated, our attention was next turned to the arrivals which had occurred from ports which were open to the suspicion of being infected with cholera.

It was found that from December 1, 1872, to July 1, 1873, there arrived from such ports 342 vessels of all kinds. The crews of these vessels numbered 7,249, and they landed 4,249 passengers—a grand total of 11,498 individuals from European, West Indian, and South American ports.

By an examination of the records of the Commissioners of Emigration for the State of Louisiana, it was found that during the same period 3,912 emigrants had arrived, of whom 1,595 were from Liverpool, 681 were from Hamburg, 1,361 from Bremen, 2 from Barcelona, 3 from Marseilles, 14

from Bordeaux, 72 from Havana, 61 from Vera Cruz, 122 from Palermo. During the year ending December 31, 1873, there arrived at New Orleans 6,079 foreign emigrants, of whom 2,938 were from Bremen, 575 from Hamburg, 1,834 from Liverpool, 239 from Havre, and 210 from West Indian and South American ports. The remaining 134 were from Marseilles, Palermo, Messina, and Genoa.

It is an established fact that prior to this outbreak in America cholera was epidemic in European Russia, Germany, Poland, Hungary, and Austria.

That the disease was again upon its westward march, and that the shores of America were again threatened, had been demonstrated in 1871, by the arrival at Halifax of the steamship Franklyn from Stettin for New York. This vessel left Stettin October 10 with 600 German emigrants on board, touched at Copenhagen and Christiansund. Eight days after leaving the last-named port cholera broke out amongst the emigrants, among whom 40 deaths occurred before reaching Halifax. The fact that she was infected was kept secret until her arrival at New York. A man who was employed watering the vessel at Halifax died of cholera. The infection was stamped out. A man who was employed coaling this vessel died at his home at Chezzetcook, and four other cases occurred in his family. At this point stringent sanitary measures were adopted, and no other cases occurred.

On the 23rd of September 1872, a coolie ship arrived at Bluff's Bay, Jamaica. It was reported that some 60 of the coolies died on the passage, and that one case of cholera had occurred upon the island among them after their arrival.

Macnamara, in his *History of Asiatic Cholera* (p. 362) throws further light upon this subject, which he obtains from the reports of the Government Emigration Agent in Calcutta.

1. That the *Sea Queen,* which arrived in Jamaica December 1872, had cholera deaths upon her during the voyage.

2. That the *Humber,* which arrived at that island in September 1872, also had cholera deaths on board during the voyage.

3. In vessels containing coolies sailing from Calcutta for Demerara during 1872, there had been severe outbursts of cholera on board the *Poona* and the *Kate Killock.* The former lost seventeen coolies from cholera between the thirteenth and fifteenth days after leaving Calcutta. The latter lost fourteen coolies from cholera between the eighth and fifteenth days after leaving Calcutta. The list of vessels

that arrived at the port of New Orleans during January 1873, shows that direct communication was established between that city and Jamaica and the adjacent islands.

The facts which have thus far been obtained go to prove—

1. That prior to the outbreak of cholera at New Orleans, Louisiana, the disease had been imported by coolie ships into the West India Islands, between which islands and the port of New Orleans a free communication was established.

2. That the Mississippi river quarantine during the last months of 1872 and the early months of 1873 was utterly worthless.

3. That during the months named a large number of emigrants arrived at the city of New Orleans from cholera-infected districts. It was endeavoured to trace the emigrants after they arrived at New Orleans.

The Commissioners of Emigration exercise no control over emigrants who arrive at that port, other than of an advisary nature. The effects of emigrants are inspected on shipboard by a Customs Officer, and upon landing the emigrant follows unmolested his own inclinations. The majority of the emigrants who arrived at the port of New Orleans in the month of December 1872, went at once to the State of Texas; but during the succeeding months large numbers passed into the interior of the United States by the Mississippi river route.

Emigrants who desire to pass northward by the Mississippi river remain sometimes for days in the city, and *lodge at emigrant boarding houses, which face the river upon the steamboat levee. It is an ascertained fact that during the months of January, February, and March, many of these houses were filled with individuals of this class.*

We were able circumstantially to trace individuals from cholera-infected districts directly to that locality of the city of New Orleans at which the health authorities of that city have told us the earlier cases of the disease became infected. The evidence, to be sure, is but circumstantial, yet upon much less direct evidence the law is justified in taking human life. The evidence is certainly strong that the unfortunate individuals who became infected with cholera upon or near the steamboat levee came in contact with the poison of the disease, which had been imported in the effects of emigrants from localities known to be infected with the disease. This hypothesis is certainly more reasonable, more in accordance with history, than it is to suppose that a *de novo* development of epidemic cholera occurred at the city of New Orleans. In view of these facts the doctrine of non-importation will not stand.

In consequence of a mechanical accident upon page 104 of the *History of the Cholera Epidemic of* 1873 *in the United States,* this investigation by Netten Radcliffe, Macnamara, and it may be others, has been attributed to a gentleman who kindly furnished us with the results he obtained from a series of interviews with the most prominent medical men of New Orleans. It is here necessary for us to claim as our own this work, be it good or bad; be its results to the benefit of humanity, or worthless and without point.

While we failed in positively establishing the method by which the disease was introduced into the city of New Orleans, we were successful in obtaining at other points positive evidence, which adds greatly to the strength of the chain of circumstantial evidence forged at that city. To them we would ask attention.

1. On the 31st of May a party consisting of Tent Have, his wife, five children, the sister of his wife, and two young men, left Tubbergen, Holland, for the United States. They were shipped from Rotterdam to Liverpool, thence to the United States. [Tubbergen is a small town of Overyssel, but a few miles (less than five) from the Hanover border. In 1873 Hanover, with many other Prussian states, was infected with cholera.] This party left Liverpool on the steamship *City of Limerick,* and arrived at the port of New York July 5th. As this family had relations at Carthage—a small town ten miles north of Cincinnati, Ohio, and desired to at once proceed there, they were forwarded *viâ* Baltimore, Md., and the Baltimore and Ohio Railroad to Cincinnati, Ohio, where they arrived on the 9th of July. At that date a mild epidemic of cholera was in existence at the last named city. But the travellers arrived late at night, proceeded at once to the Dayton Railroad Depôt, where they passed the few remaining hours of the night, and early the next day proceeded to Carthage. Arriving at Carthage, the men, with the characteristic industry of their nation, at once found employment at the gravel pits. The women and children rested, awaiting the arrival of their effects.

On July 13th, the boxes and bales of household property having arrived, the family took possession of a house. Their effects, which consisted chiefly of bedding and woollen clothing, were unpacked and hung up in and around the house to dry after the long voyage; and it is asserted by the survivors that this had not been unpacked since leaving home.

On the 15th of July a male child three years old was attacked with cholera. This occurred ten days after landing

in New York city, five days after their arrival at Carthage, and within forty-eight hours after the unpacking of the chests, and the exposure of their contents to the sun and air. The same day the father, who had been called from his work by the illness of his child, and was directed by the attending physician to cut down and remove some rank weeds growing around the house, was taken with the same disease, and died within eight hours. At almost the same hour that the father was taken sick a second child five years of age was attacked, and lived only twenty hours.

The next day (July 16th) the mother sickened and died in twelve hours. The next day (July 17th) a third child, seven years old, died after fifteen hours. A lapse of one day, and a fourth child, eight years old, died after twenty-four hours' illness. July 22nd the fifth child, four years old, died, after an illness of twenty-four hours, and the next day the young woman died also. The two young men suffered severely with diarrhœa, but in neither did the disease advance to the second stage.

Under the direction and supervision of the attending physician, the excreta of all these cases were disinfected with sulphate of iron and buried, and all the soiled articles of clothing that could be discovered were burned. The most active system of disinfection was instituted in the village, and but one case occurred outside the house occupied by the Tent Have family.

On July 23rd, the day of the last Tent Have death, a young woman, married, and pregnant with her first child, and who lived in a comfortable house about one hundred yards in the rear of the infected house, was taken with diarrhœa. On the 25th, cholera was fully developed, and she died after forty-eight hours' illness, having aborted a four months fœtus some twenty hours before death. It was subsequently discovered that this lady, although forbidden by her husband to go near the infected house, had prepared articles of diet for the sick, which she each day carried to the fence in the rear of the house to which the cholera was confined. Surely such charity covereth a multitude of sins.

No other cases occurred in the town of Carthage; but, on the 21st of July, the day upon which it will be remembered the last child of the Tent Have family was ill with cholera, the disease suddenly appeared at the Longview Asylum, in the negro department, and in the person of a female who was employed as the cook. The case was isolated; the excreta were disinfected with sulphate of

iron. The patient lingered until the 23rd, when she died. No other case occurred until July 26th, when a white patient was attacked, and the same day four other cases occurred. July 27th, two more cases occurred. These eight cases all terminated fatally. July 28th, a case occurred, which recovered; July 29th, a fatal case is reported, and three cases which recovered.

It has been asserted that in no way could a connection be established between the cases at the asylum and those in town; that no communication existed between the asylum and the infected house; therefore, the origin in the asylum must be considered as a distinct demonstration of the disease.

The negro department of Longview Asylum is in a large house situated at some distance from the main buildings of the asylum. It is a large brick building upon the western extremity of the asylum grounds. In front of this building pass the canal and the Dayton Short-line Railroad, and, in the rear, a street to and from the village. The insane inmates of this house were isolated from contact personally with the cholera sick, but no restrictions were adopted by the attendants, who were allowed all their usual liberty; and, actuated by humanity, the physician in charge of the negro division of the asylum had visited in consultation the cholera sick.

The introduction of the disease into the asylum, however, is not to be left to any conjecture; for it is asserted that Joseph Marshall, the supervisor of attendants in the negro division, frequently went from curiosity into the house where the Hollanders were ill of cholera, and that from this house he time and again returned to the asylum. A morbid curiosity seemed to possess this man; he could not resist the impulse; and, as often as he could frame an excuse for leaving the asylum, he visited the infected house. This man Marshall, who is a negro, passed a portion of each day in the asylum kitchen, and in the company of Delila Pointer, the first of the asylum cases. Marshall was at the Tent Have house when the last of the family died, and, as they were all dead, he could see no reason for his not appropriating a curiously braided coat which had belonged to the dead Tent Have. The negro love for finery overcame any scruples he may have had; the coat was concealed and carried to the asylum, when, as it was found to be wet, it was hung up on the back porch to dry. While in this position it attracted the attention of a patient named Howard Preston, who, putting on the coat, wore it for a day and slept in it one night, when it was again taken possession

of by Marshall. The next day Preston and four other inmates of the asylum were taken with cholera, when Marshall, properly connecting the outbreak of the disease with the stolen coat, destroyed it at the kitchen fire. These facts were obtained by voluntary confession.

At Longview, thirteen cases of cholera occurred, nine of which terminated fatally; of these cases, nine were females, four were males. Six females and three males died. Of the total number of cases, six were whites, seven were negroes. Five whites and four negroes died. The occurrence of white cases at the negro division is accounted for from the fact that, some time prior to the outbreak of cholera, owing to the crowded condition of the wards in the main buildings, a number of demented whites had been placed in the negro division, where they were mixed with the other inmates. The main buildings of the asylum were isolated from the infection, the attendants rigidly respecting the orders issued by the superintendent. The medical gentlemen who, as in duty bound, met the call for professional advice, adopted every precaution to avoid the introduction of the disease into the asylum, and successfully, for no cases occurred in the main buildings where these gentlemen reside.

2. A family named Antonson, consisting of a man, his wife, and four children, with a young man named Oleson, left their home at Alfoden, Sweden, for the United States. They travelled to Bergen, some two hundred miles. At that port they remained three weeks, awaiting the departure of a steamer, and, finally took passage for New York on the *Peter Japson*. They arrived June 26th, 1873.

From New York City the Antonson party were transported to Grand Haven, Michigan, remaining but one hal hour at Pittsburgh, Pennsylvania. At Grand Haven they remained one half day and one night, and thence proceeded to Saint Paul, Minn., where they rested twenty-four hours; thence to Crow River settlement in Kandiyolii county, Minnesota, *viâ* Willmar.

On July 2nd, on arriving at Willmar, and preparatory to a wagon ride of forty miles, as the weather was very warm, the boxes of personal property, which had not been touched since leaving the Baltic, were opened, and lighter clothing was taken into use. The next day, Oleson sickened with a diarrhœa, cholera was developed, and he died July 10th. Three of the children of Antonson died in quick succession. Next Errickson, at whose house the party had put up, died. The wife of Antonson and his remaining child had the dis-

ease, but recovered. All the inmates of the house at which these deaths occurred suffered with diarrhœa, and three deaths are reported among persons living at a distance, but who had visited the house during the illness of Oleson. After landing in the United States, this family had passed through no cholera-infected territory or city. They were taken sick with the disease in a previously healthy country far removed from all specific causes of the disease previous to their arrival. The members of this party were the first sufferers from the disease; but they brought death from the Baltic sea, not only for themselves but for their hosts.

3. During the years 1872 and 1873 large numbers of Russian emigrants reached the United Siates. They arrived in companies of from ten to twenty families, each family having an average of eight children. The majority of these people came from Odessa and the Crimean districts. Each band was a community headed by one man who was selected to assume the responsibilities of his party. Some few were persons of wealth and respectability, but the large majority were of the lower classes, filthy in their persons and habits. Each family brought with them boxes and bundles of their personal effects, which consisted of articles of clothing, bed-furniture, and cooking utensils. In the boxes which contained the articles for use upon the journey, articles of clothing, and those of food, were generally to be found in an undivided chest.

Upon their arrival at Yankton, these people were cared for by the citizens of the city. They were given quarters in unoccupied buildings, supplied with food, and every effort was made to render them comfortable until such time as they could be located upon the farms in the territory that had been selected for them. It was observed that in the majority of instances it was impossible to compel them to adopt any sanitary precautions in their lives; the utmost repugnance was shown to the use of privies, both sexes preferring to urinate and defecate immediately around the building in which they were located. On the 25th of August, immediately upon the arrival of a party of these Russians, who had reached Yankton immediately from New York, two cases of cholera occurred in the persons of children, who, with their parents and several other families, were lodged in an unoccupied school-house. These cases terminated fatally, after a few hours' illness. The next day five other cases occurred in the same room, and nine others in an adjoining room. Of these fourteen cases but two recovered. From these cases the disease spread, and the

epidemic continued in force until about the 15th of September, when its epidemic influence disappeared. From August 25th to September 15th, forty-two cases of cholera occurred among the bands of emigrants. Of these, twenty-nine were fatal. The large majority occurred among children of less than sixteen years of age. Comparatively but few deaths occurred among the adults. In this connection it must be borne in mind that over two-thirds of the entire body of emigrants were minors.

After the development of this disease among the Russians, a few well-defined cases of cholera occurred among the residents of Yankton, while at the same time diarrhœal diseases were unusually frequent. Early in September, a lot at the corner of Third Street and Broadway was cleared for the purpose of erecting a block of buildings. The dirt and *débris* that were removed from this site were used to fill some inequalities on Third Street. Among other refuse removed was the contents of an old privy, which had been used by some Russians who had been quartered in an adjoining building. This privy soil was scattered over the street, to secure its rapid oxidization; but, in so doing the stench was unendurable.

On September 5th, a Mr. M., whose business called him many times each day to pass over this ground, was attacked with cholera, and died after a few hours' illness. The next day his wife was attacked with the same disease, but recovered after a severe illness. In these cases a thorough system of disinfection was instituted; the bed, bedding, and soiled clothing were burned.

On September 6th, a man named McUmber, who was working upon some new buildings in the vicinity of the point on Third Street upon which the *débris* from the privy-vault had been scattered, was taken with cholera and died after a few hours' illness. The same active system of disinfection was instituted. The same day an Indian, who had pitched his tent immediately adjoining the house in which McUmber was sick, was attacked, but recovered after a serious illness.

On September 7th, a man named Faust, who lived fourteen miles north of Yankton, and who had been in the city on the previous day, was taken with cholera, and died after an illness of about ten hours. This man was attended by some friends who had been through the cholera epidemic of 1866 and 1867. By them everything was carefully disinfected. The wife of Faust and his child were removed from the house before his death. After death, sulphate of iron in quantity was placed in the coffin under the body. His bed, bedding, and clothing were destroyed by fire.

On September 9th, a Mr. Presho, who lived in close proximity to the polluted point upon Third Street, was taken with cholera, but ultimately recovered.

On September 14th, a little girl, whose family resided in the district in which the Russians were lodged, and directly opposite a house occupied by a number who were intensely filthy in their habits, was taken with cholera, and died after an illness of six hours. The next day, a second child in the same family was attacked, but recovered. The cases were followed by six well-marked cholera cases in the city, all of whom recovered; a total of twelve cases, with four deaths.

After the removal of the Russians from the city to the farms in the counties of Yankton, Bonhomme, Hutchinson, and Turner, no accounts can be obtained. That fatal cases still occurred there is no doubt; but as no American physician was called in, it is impossible to bring them to light.

During the month of November 1874, an inspection of the cholera-infected districts of 1873, in Dakota Territory, was made by the writer. A most careful and patient research among the emigrants failed to elicit any new information. By some of intelligence, it was admitted that the districts in Russia from which they had departed were cholera-infected, and many instances corroborative of published reports as to prevalence of this disease in the Southern provinces of Russia were obtained. One man of much intelligence and refinement recounted interestingly the frequent arrivals of the disease at the city of Odessa, and another man from the vicinity of the city of Taganrog, confirmed the cholera-reports published from the Crimea during the past few years. But one and all were interested in concealing the events among them, as far as the influence of cholera was concerned after their arrival in the United States. Had it not been that the physicians of the city were active in their observation of the disease, an extended outbreak would have occurred.

A personal inspection of these people will demonstrate that they are peculiarly liable to become the porters of disease. In their persons and in their habits they are filthy. The use of water is almost unknown among them, and when personally used is, in the majority of instances, perverted. A gentleman of Yankton, who was much interested in the study of these people, informs us that upon one occasion he observed a female wash her hands, head, feet, and other portions of her body in a pan of water. Her ablutions having been accomplished, she proceeded to wash a cooking-utensil in the same water, and, finally, before it was dis-

charged from use, some meat and potatoes, upon which in a short time the noon-day meal of her family was made.

The clothing of these people consists of heavy, coarse, woollen fabrics. Their beds are generally of feathers, their blankets thick and heavy. The men wear a great-coat of the shape and size of an Ulster, made of dressed sheep-skins with the wool retained. Their persons are unkempt, their hair and beards long. The adult male is the only individual of the family of importance. The children, alive or dead, are noticed but by the mother, who, besides her household duties, performs the labour of a field-hand.

4. A significant case, and one strongly corroborating the chain of circumstantial evidence, is found in the narrative of the epidemic of 1873, as it affected the city of Chicago, Illinois.

The first case that occurred in that city was on May 24th, in a person recently returned to his home from Memphis, Tennessee, at that time a cholera-infected city.

The second case occurred June 10th, at a point fully two miles from the first case. The disease now occurred in a house occupied by several families of Danes. At this house a Dane, who had lately arrived in this country by way of New Orleans (where he had been ill) had been sick with what was supposed to be typhoid fever, but from which he recovered. While he was in this typhoid condition the first cholera case occurred, followed soon by a second. The third case occurred in an opposite house, in the person of a woman who had helped to care for the first case; and from this point, as a focus, the disease spread.

The stranger recently arrived was evidently the exciting cause of the outbreak. As soon as he was sufficiently recovered he left the city and could not be traced.

These instances are presented as strong links in the chain of circumstantial evidence advanced, that the diffusion of cholera to the Western world in 1873 presented absolutely no differences from the known methods by which the disease has always advanced in its march through the world. *To human agency alone can it be ascribed.*

By the arrival of a family from cholera-infected Europe the disease was developed in an inland town many hundreds of miles from the seaboard. This family, after landing in the United States, had passed through no infected localities until they reached the city of Cincinnati. But this city they did not reach until a late hour of the night, and then did not go to any lodging house, but remained in a depôt building in a portion of the city that had presented no evidence

of infection. It is very true that it may be claimed that, as the disease was epidemic at Cincinnati, it may have been carried to Carthage in some other manner; but cholera appeared in Cincinnati on the 27th of May, and the Carthage cases did not occur until July 15th. The disease did not then occur upon the arrival of this family, but after the arrival of *their effects*. Let it be remembered that although "Longview" is the asylum of the county, although it is in daily communication with Cincinnati, yet the institution was wholly free from the disease until after the coat of Tent Have had been carried within the walls.

But the Minnesota cases are free from all these objections. They journeyed through an entirely uninfected portion of the country, and no point through which they passed was infected before their arrival or after they had passed, save the city of Pittsburgh, Penn. At Pittsburgh a group of but four cases occurred over a month after the Antonson family had passed through the city, and these four cases are most distinctly traced to the epidemic in the State of Ohio. If the Antonson party did not bring cholera in their effects into the wonderfully healthy regions of Minnesota, *a locust cloud must have struck an aerial wall or something*, and have swarmed down upon the Crow river settlement just as they arrived.

As to the Russians, it is very true that after reaching the United States they were obliged to travel through cholera-infected territory. But their transit was rapid. Their ignorance of the language of the country held them together. There was no straggling from their companies. Their suspicious nature would have led them to repel all advances of strangers. As their emigration was an organised movement, the man who was selected to command each band was in almost every instance one who had been sent out by former emigrants to especially act as a convoy to the strangers.

Be it as it may, one fact remains absolute. These people were absolutely free from the disease until they had unpacked their effects and commenced to establish themselves in their new home.

NOTE ON MEDICAL STATISTICS IN THE PUBLIC HEALTH SECTION OF THE NINTH INTERNATIONAL STATISTICAL CONGRESS, HELD IN BUDA-PESTH, HUNGARY, IN SEPTEMBER 1876.

By F. J. MOUAT, M.D.,

Foreign Secretary, Statistical Society.

(*Read: May 9th*, 1877.)

In the preface to the separate publication of the paper on medical statistics, which I was privileged to read before the Epidemiological Society in 1872, I advocated the institution of a special biological section of the International Statistical Congress, for the consideration of all questions of life and health in their strictly statistical relations.

In it I stated that "it cannot be contested that questions concerning the life, health, growth, development, decline, and decay of man in his mental and physical relations, are of at least equal importance with matters relating to his social and political state. The movements of commerce, the administration of justice, legislative measures generally, and all that concerns the well being of society as a civilised community, are objects of statistical inquiry of which it would be difficult to over-estimate the interest and importance."

"And yet does any one of them, or do they all together, touch more closely the happiness and well being of man as an individual entity than those which affect him personally from the cradle to the grave?"

"The late honoured and lamented M. Quetelet has indicated generally the direction which such investigations should take. There is little prospect of their receiving the attention they demand, unless the consideration of biological questions is entrusted to a separate and distinct section of the congress."

"To it should be assigned census returns, statistics of births, marriages, and deaths, the statistics of disease in its therapeutical and pathological relations, so far as they can

be subjected to arithmetical tests; the statistics of insanity, the determination of all questions of life assurance in the more extended relations which the movements of population and commerce now demand; the statistics of hospitals, and, in general, all that relates to public health in the widest sense, to which the numerical method is applicable, and in which its rigorous exactness is so much needed."

"In the public discussions on almost all practical questions bearing on health and disease, such as hospital construction, the disposal of sewage in its hygienic relations, the causes of epidemic outbreaks of disease, and many similar matters, it is painful to witness the groping in the dark, and the widely divergent conclusions at which even observers of eminence arrive, from the absence of any thoroughly reliable foundation of fact on which to construct measures of prevention or removal. Statistics alone cannot solve such questions; but they can clear the way for their scientific consideration, and by determining the exact cause of an evil, lead by strictly inductive processes to the measures likely to be efficacious in removing it or mitigating its effects."

The Permanent Commission, to whom this appeal for a separate section was addressed, did not take it into consideration, and the Congress of St. Petersburg had left the question in a doubtful state, by expressing a hope that a sufficient number of medical men would attend the next Congress, to render it possible to constitute a separate section of Medical Statistics. As this could not be ascertained prior to the assembly of the Congress itself, the Hungarian organizing or local Committee prepared the materials for the consideration of such a section should it be formed, and two of their number, Professors Fodor and Korànì, drew up reports for consideration on a system of international statistics of epidemic diseases and of cholera.

The international statistics of baths and mineral waters was also discussed, but as they have no reference to the special objects of the Epidemiological Society, I shall not refer further to them.

The Russian Physicians of St. Petersburg had promised to submit to the succeeding Congress the results of the application of the method of inquiry proposed by them with respect to the study of Cholera in Russia, but failed to do so, without reason assigned.

Germany sent a large contingent of Doctors to the Congress, but as it is difficult to determine to what variety of German Doctorate they belonged, I cannot say how many

of them were members of the Medical Profession. Hungary had naturally many medical representatives, the Congress having met in the capital of that country, and the rest of Europe sent some half dozen. England was represented by Dr. Farr and myself. It is manifest that the profession which has to deal immediately with public health in its technical aspect, was in no fitting sense represented at the Congress, and I am afraid that the time has not yet arrived when Medical questions have a chance of being discussed in a strictly scientific manner at any General Congress. I am, therefore, *now* of opinion, that the Public Health Section of the International Statistical Congress had better be suppressed, at any rate for the present, and that a special statistical branch should be added to the Medical Congresses now in existence.

Among the questions which press most urgently for solution is that, for example, of a proper system of hospital record to determine a number of questions which demand the scientific application of numbers for their solution. The statistics heretofore collected are constructed on far too narrow a basis to afford more than a very rough, and in some instances, probably inaccurate idea of the risks to life from treatment in particular public institutions. Except in regard to amputations, fevers, parturition, and such diseases as are treated in special institutions, the returns afford no information whatever of a multitude of facts of vital importance in relation to disease generally, which are scarcely of less interest than a more accurate and extended acquaintance with epidemic visitations. And even as respects those diseases which *have* been the subject of special investigation, the records are not so specific as they ought to be. In fact, the remarks on this subject made by M. Husson in 1862, in his great work entitled *Etude sur les Hôpitaux*, are as true now as when they were written.

In the eleventh section, under the head of the Medical Statistics of hospitals, this great and sagacious administrator remarks:—

"After an attentive study of all the material ameliorations which the administration has introduced into the service of these hospitals, it remains to examine the results attained by so many efforts, and so much solicitude for the care of the sick.

"Figures, vigorously exact, can alone enlighten us upon this point; but it is, unfortunately, only too certain that beyond the tables of mortality, published annually, and which present, in an abstract form, the relation of the

number of admissions to that of deaths, the hospitals of Paris have never possessed the essential and indispensable elements of a rational and conclusive system of statistics.

"It is true that since 1837, the Administration has gathered together from each hospital an account of their great surgical operations and their results. But this is only a special side of the question, and with regard to it, we shall see anon how defective these statements, the object of a special examination of a particular subject, really are.

"It should be, then, with the greatest reserve that the tables published by the Administration since 1804 must be studied to ascertain the death-rate in the hospitals, and the average duration of the treatment.

"These documents can be studied with profit only on the understanding that they contain general facts and summaries from which the light of circumstantial details is absent."*

The Third Section of the Congress held in the Capital of Hungary was devoted to Public Health, under the Presidency of Dr. Rapp, a Royal Councillor and Professor in the University, with Drs. Farr, Hirsch, Janssens, as Vice-Presidents, and MM. Grosz and Balagh as Secretaries.

A private meeting of the Medical Members of the Section was held at the house of Professor Korángi, to consider the procedure most desirable to adopt at the public meetings of the Section, and to regulate the proceedings generally. As customary on such occasions, the time allowed for discussion was not sufficient for much more than verbal criticisms; and as there were only shorthand writers for French and Germans allowed, there is no report of what fell from the English Members. The discussions, as a rule, were of little real value or interest, and as these matters are at present conducted, it cannot well be otherwise.

The international importance of collecting exact and detailed information regarding the origin, extension, and results of epidemic diseases over as wide a field as possible having been duly considered, as well in their general relations as with special reference to Cholera, the following resolutions were recorded by this Public Health Section.

"(*a.*) Propositions regarding the Statistics of Epidemic Diseases in general.

"1. The Congress considers it a proper function of International Statistics to study epidemic diseases, in the interests both of science and of public health.

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* *Etude sur les Hôpitaux*, par M. Armand Husson. 4to. Paris, 1862, pp. 247-8.

"2. International Statistical Inquiry should above all take note of epidemics, local and accidental (temporary) circumstances, and the degree of intensity of the most important epidemic diseases, such as cholera, plague, yellow fever, typhoid fever, typhus, dysentery, influenza, diphtheria, croup, inflammation of the respiratory organs, small-pox, scarlet fever, and whooping cough."

To effect this object the Congress proposes that:—

"3. Every State, participating in International work is bound to promulgate monthly a report containing the following information:—

a. In what arrondissements (or other corresponding administrative units) and in what towns there has appeared during the preceding month any epidemic.

b. The nature of the epidemic in question.

c. Its death rate.

"4. One of the chief Statistical Offices should be charged with the organization of an International Statistical Branch for epidemic diseases, and to this branch office the bureaux of different States should be enjoined to transmit as soon as possible (by telegraph), the facts relating to epidemics, founded on which the Central Office should issue a monthly statement including the points above-mentioned. These publications should be sent to all States taking part in International work, so as to become accessible to scientific men, and even to the public.

"5. The cost of this work to be divided among the States co-operating in International work.

"6. The Permanent Commission of the Congress is charged with the execution of this resolution, and with the elaboration of such details as have not been definitively determined."

Stripped of its obscurity and verbiage, the meaning of this resolution seems to be that every nation shall take immediate note of the appearance of epidemic diseases, shall communicate the fact to all other nations; and shall take steps to put this information into a shape fit for publication and communication to all the world at short periods.

That so much of this as is of immediate international importance should be carried into effect, I hold to be both practicable and desirable. With regard to the greater number of diseases mentioned, however, it is wholly unnecessary, would incur a vast deal of fruitless cost, and be of no immediate use in an *international* sense.

If the action were restricted to cholera, plague, yellow fever, small-pox, and possibly influenza, it would accomplish all that is of immediate use; and even with respect to those diseases, the communication of the fact and immediate circumstances of an outbreak would direct attention to the matter; which is, after all, the only essential point of strictly immediate international importance.

The scientific study of the phenomena of each particular outbreak is not a matter which can be touched by telegraphy. It demands time, care, patient study, the minute scrutiny and analysis of all facts calculated to throw light on the origin, mode of extension, proper treatment (curative and prophylactic), and stamping out of the disease, should

such a proceeding be practicable. All this should undoubtedly be collected with the utmost patience and minuteness, and after a careful digest, be published by each Government annually in a form fit for general distribution. The pouring out of floods of figures, assumed to be accurate exponents of facts, which they very seldom are, is an unmixed evil, and does more harm than good. This is the cardinal error of the Indian Sanitary returns and reports, valuable as they are in some respects. They are a crude mass of ill-digested and undigested numbers, which few have time to wade through, and fewer still know how to deal with analytically; and they certainly give sanction to the apothegm of one of the Fathers of Medical Philosophy—that there are more false facts than false theories in Medicine.

It should be the duty of those who collect the facts and figures to digest them, and to formulate the conclusions which they appear fairly to establish, in a concise and intelligible form. The figures themselves should be published as a guarantee of the good faith of the reporter, and to enable others to verify his deductions, should such a proceeding be deemed necessary.

I myself should look with grave apprehension to the adoption in their integrity of the proposals of the Health Section of the last International Statistical Congress. Fortunately, there is no chance of their acceptance with us, inasmuch as it would be difficult, probably impossible, to persuade the Parliament of Great Britain, with whom the matter really rests, of the necessity of the outlay that would be required to carry them into effect. They would inundate us still further with figures of little practical value, and, by introducing confusion worse confounded, certainly not tend to advance our knowledge of the very important matters to which they relate.

Again, it would be much simpler, and more satisfactory, that each Country should pay its own cost, and not attempt to have a running account with other nations which would be difficult to adjust.

The fact of an outbreak of epidemic disease, and a brief account of the exact circumstances in which it arose, ought certainly to be communicated at once by telegraph to all other nations. This would enable each State to determine what special measures, if any, were necessary to prevent its introduction or spread, whether by human intercourse, merchandise, or any of the other ways in which such diseases are known, or believed to be propagated. The telegraphic action should afterwards be restricted to brief notices at

short intervals of the increase, diminution, decline, or disappearance of the disease.

The subsequent investigation of the exact cause of each epidemic should be the work of the country in which the outbreak occurs, and the inquiry should be conducted on some uniform plan, so as to render the results comparable for international purposes. For this purpose I know of no better plan than that suggested by the Statistical Congress held in Paris in 1855. It is somewhat too minute and detailed for practicable purposes, but the general outline of the scheme contains all that is really necessary for strictly international ends.

The next question considered was that of Cholera, in which the views enunciated were evidently tinged with the hues of the Congresses of Constantinople and Vienna, neither of which bodies appear to me to have so treated the question as to deserve to be accepted as conclusive authorities.

The resolutions of the Statistical Congress respecting Cholera were so minute, detailed, and full of particulars of no special interest or value in throwing light upon the disease, that I do not think it necessary to trouble you with them, as I am quite certain that they are thoroughly unworkable. They are manifestly based on those of St. Petersburg, but encumbered with an amount of additional minutiæ that would seriously embarrass, if they did not entirely overwhelm, those who had to digest them, without any corresponding advantage. For example, regarding each individual attacked the following particulars are required, viz.: his name, age, religion, family status, occupation, quarter of the town in which he resided, the street, number of the house, floor, and number of the lodging in which he was attacked; his constitution and physical state at the time of attack; any extraordinary circumstances regarding him which preceded the attack, such as his manner of life, work, food, individual health, emotion, fright, moral pain, physical effort, mental tension, with a great deal more of solemn trifling, which it would be impossible to ascertain, and which would not be worth recording if it could be obtained.

Then the bulletins are to be furnished on paper of different colours, and divided in coupons according to a fixed formulary; yellow for houses in which there were more than two cases of cholera; blue for houses in which there were only one or two cases; and green for houses in which there were no cases.

You are aware that I am not an enemy to the collection of all details really necessary to throw light upon the origin, course, extension, and effects of this, or any other epidemic disease; but these should not be overladen with particulars of which the necessity is more than questionable, or which would demand an amount of inquisition impossible to exact in the harass and anxiety attendant upon the presence of such diseases in a household or a community.

Moreover, I am by no means convinced of the expediency or wisdom of placing Cholera in a category of its own, and separating it so entirely from all other epidemics, as the labours of what I cannot but consider the abortive Congresses of Constantinople and Vienna would seem to advocate. Some of the errors of the former were not repeated in the latter, but neither of them have indicated the true solution of the question. All epidemics doubtless obey laws which it is desirable to ascertain, but most of them have certain features in common which show that a similar method of inquiry applied to all would be attended with advantage. Anything that is absolutely special may by an additional column or two be obtained: beyond this, all minute subdivisions and special modes of investigation, complicate without facilitating the collection of the data necessary to their right understanding.

As respects Cholera, I am strongly of opinion that it can best be studied in the country in some part of which it is always present, and that it is useless to rely upon European Congresses to determine the right method of dealing with it. So far as my personal experience goes, and I believe that I have seen as much of it as any living physician, no theory yet broached will explain all the phenomena connected with it. Occasionally, each and all of them break down—hence the urgent necessity of collecting the facts with care and discrimination, so as gradually to eliminate all that is accidental, temporary, or due to purely local causes, and to determine those conditions, should there be any, which are never absent, and which may be rightly regarded as pathognomonic.

Many years ago, in 1855, I strongly urged the Government, under which I was serving, to permit me to collect exact data of Cholera as it appeared in the prisons under my charge, and in 1857 I framed rules for the Medical duties of the prisons of Lower Bengal. Among them was a special Cholera Register. This was carefully kept for six months, and promised to become most valuable, when the Medical Authorities interfered, would neither permit me to collect

the data, nor do so themselves, and so I was compelled to give it up, as the Medical charge of the prisons was not in my hands.

To return however to the Congress, the chief point of difference with the St. Petersburg proceedings was in the addition of a special section for the statistics of cholera in relation to shipping and ports. This, again, is so overladen with details, of which it is difficult to see the use, that I shall not trouble you with them, as I regard them as thoroughly unworkable. I have myself been in quarantine, and on board vessels which had to produce bills of health, and obtain free entry, or be subjected to quarantine; and anything more thoroughly perfunctory and useless than the whole proceeding it has never been my lot to witness. On one occasion, the foul bill of health carried was deliberately destroyed, but I was able to explain to the port surgeon the absolute absence of risk from the vessel and its tenants, and the governor of the colony sent us speedily a free pass. On another occasion, when travelling from Alexandria (where a most disgraceful and contemptible cholera panic existed) to Trieste, a gentleman was attacked by what the Polish surgeon of the ship considered to be cholera, and thereby nearly frightened the patient and the passengers in the Austrian Lloyds' steamer to death. He had administered five drops of laudanum in a cup of tea, which he considered an alarming dose, and applied a rag dipped in aromatic vinegar to his forehead. At the urgent entreaty of his friends, I was asked to see him in the middle of the night, and found him to be suffering from violent colic, from overloading his stomach at Corfu, after three days' compulsory starvation from sea-sickness. A dose of castor-oil dissipated the cholera and the panic, but nothing could convince the good gentleman that I had not saved his life from a terrible scourge, and the port sanitary authorities looked upon us as an unusually suspicious lot, and gave us an additional dose of quarantine in consequence. The arrangements of the lazaretto in which we were confined were of the most incomplete description, so that, had cholera really been a contagious disease, in the sense that scarlet and typhus fevers are contagious, we could not have failed to have introduced it to the port of Trieste. The manner in which the quarantine was carried out was childish in the extreme, and I have no doubt whatever that in all cases where such regulations cannot be enforced so as to render the isolation absolutely complete, they are a vexatious interference with the movements of commerce and human intercourse, without any real

protection to those whom they are intended to shield. The errors of the Constantinople Conference have been very mischievous in the influence which they appear to have exercised on most European nations, and it is somewhat disheartening to find them pervading the provisions of a convention subsequently drawn up in Vienna, to which I shall refer more particularly anon.

When the Government of India wished to establish sanitary cordons and to institute a land quarantine, after the great cholera outbreak at Hurdwar, upon the recommendation of a lay sanitary commissioner of great ability and eminent administrative capacity, but who had no technical or professional knowledge of the subject, I remonstrated strongly against it in a paper which I lay on your table. I had hoped that the phantom and delusion of the efficacy of quarantine had taken their place with the Cock Lane Ghost and other exploded fallacies, but, as it still appears to have occasional fits of vitality, like the great Sea Serpent, it is as well to draw attention to the abundant evidence in existence of its utter uselessness, from the impossibility of carrying it out completely.

The draft of an International Sanitary Convention was drawn up shortly after the Cholera Conference at Vienna, to regulate measures for the prevention of the extension of that disease. This Convention consisted of thirty-three articles, and although it has never been ratified, so far as I know, it is an important document, and deserves to be more widely known than it is at present, for I have seen no public notice of it.

The first article is to the effect that to prevent fresh invasions of cholera in Europe, the high contracting parties approve for countries out of Europe the measures recommended by the Conference at Constantinople, and more especially quarantine in the Red and Caspian Seas. "These quarantines to be instituted and organised in a complete and satisfactory manner in accordance with the strictest maxims of hygiene."—*Non possumus.*

The second article refers to the measures to be adopted in European ports, according as a system of medical inspection or of quarantine is performed—giving to each, or to both when united, power to enforce them in certain adjacent parts of the coast, and binding them to exercise whichever system may be adopted.

The third article fixes the system of medical inspection, by providing the means and agency of such inspection at each port, according to its maritime and commercial importance.

The fourth article allows free admission to vessels which come from a clean port, have touched at no suspected port, have held communication with no suspected ship, and have had no case on board during the voyage of a suspicious character. The form of proof required to establish this is left to the determination of each high contracting power, either from a clean bill of health, the solemn declaration of the commander, or in any other way preferred. None of these are reliable.

The fifth article requires that any vessel with any suspicion about her shall be subjected to rigorous medical examination, to determine her sanitary state on arrival. As I have seen such examinations conducted, they are utter frauds.

By Article 6, if nothing compromising is detected, free pratique is to be granted. If anything suspicious should have happened during the voyage, everything shall be carefully disinfected, notwithstanding that the ship and passengers have been found free from cholera in the port.

Article 7 makes provision for dealing with suspected cases, or deaths from cholera, and the rigorous disinfection of all clothing and effects in a special place under the rigorous supervision of the sanitary authorities.

By Article 8, merchandise is to be passed free, with the exception of rags and other things capable of carrying contagion.

Articles 9, 10, 11, and 12, deal with the duration, nature, and conditions of observation and quarantine generally.

Article 13 refers specially to pilgrim, emigrant, and other ships deemed dangerous to public health.

Article 14 provides for sending an infected vessel to the nearest lazaretto, where no local means of dealing with it exist.

Article 15 requires a ship to be treated as coming from an infected port, although it had been in an intermediate port without having been subjected to quarantine.

Article 16 gives a loophole for escape from rigorous measures, in cases of mere suspicion.

Article 17 enjoins simple measures of disinfection, in ports where cholera is already epidemic, without a resort to quarantine.

Article 18 is of sufficient importance to demand reproduction in full.

"The Captain, Medical Officer, and Officers of the ship are bound to declare to the Sanitary Authority all that they know of suspicious

appearances among the crew or passengers. In case of false declarations, or of wilful reticence, they are liable to the penalties of the Sanitary Laws."

"The High Contracting Parties shall hereafter come to an understanding of the bases for the elaboration of an International Sanitary Penal Code. Recognising the necessity of such a penal sanitary law being promulgated in the Ottoman Empire, the care of framing it, and of submitting it to the approval of a special International Commission shall devolve on the Imperial Ottoman Government."

We are at least a century removed from the possibility of enacting such an International Code, and the Turkish Government is the very last to which it should be entrusted.

Article 19. The manner of carrying out disinfection is left to the judgment of the competent authorities of each country.

Article 20 abolishes river quarantines as impracticable and useless, but retains a system of medical inspection.

Article 21, for the same reason, abolishes land quarantines.

The next articles are deserving of full reproduction, as, if ever carried into effect, they will prove an epoch in the history of medicine; but one, in my belief, of very doubtful value.

Article 22. A permanent International Sanitary Commission shall be instituted at Vienna for the study of epidemic diseases.

Article 23. The functions of this Commission shall be purely scientific; it can be consulted with this view by the high contracting parties. The principal duty of this Commission shall be the study of the etiology and prophylaxis of cholera. Nevertheless, it can include in its studies other epidemic diseases. To this end, it shall frame a comprehensive programme for the investigation on an uniform plan, by all the contracting states, of the causes and treatment of cholera, and other epidemic diseases, bearing in mind generally the proposals on the subject made by the Vienna International Sanitary Conference in its deliberations on this point.

It shall make known the results of its labours by means of reports regularly submitted to the high contracting powers, and by general publications.

Finally, it can propose the convocation of International Sanitary Conferences, and will be charged with the elaboration of the programme of these Conferences.

Article 24. The Commission shall consist of medical men delegated by the high contracting powers.

Each Government or independent sanitary administration has the right of being represented by a delegate.

The Commission shall be independent as to its scientific

labours, and in consequence shall correspond directly with governments and administrations, and eventually with the special authorities indicated by the high contracting powers.

Articles 25 and 26 provide for the procedure and proceedings of the Commission under the authority of the contracting parties.

Article 27. The high contracting powers shall give to their sanitary authorities and health councils the necessary instructions to furnish the International Commission with all required information relative to questions within the circle of its studies.

Notably, the high contracting parties shall cause the first cases of epidemic cholera in their dominions to be reported, especially if they occur in the seaports of Europe, and they shall without delay instruct the Commission to collect all the data of a complete statistical record of the progress of this epidemic.

In the countries where International Sanitary Councils are established, these shall furnish all the information they possess, and shall indicate the necessary researches.

Articles 28, 29, and 30 relate to details of the working of the Commission, and the regulation of the payment of the cost of the Commission, which is not to exceed £10,000 a year.

The remaining articles also refer to details of arrangement, and to fixing the duration of the Congress at ten years.

I am not aware that this Convention has ever been ratified, so that it raises a question well fitted for discussion by this and other medical societies, viz., as to the best method of investigating the phenomena of epidemic diseases in general, and of cholera in particular, should it still be deemed expedient to separate it from other affections originating, spreading, and destroying in the same or in a similar manner. It is manifestly too large a question to discuss as an incident of my report of the Public Health Section of the last International Statistical Congress. But I may remark that I do not deem an International Sanitary Congress to be in any sense a fit and proper body to undertake such an investigation. There is no guarantee that the fittest men will be selected by each Government as its representative, or that the fittest men, if selected, would undertake the duty; and no one man, however fit, can hope to conduct such an inquiry to a successful issue. It would be far better for each Government to expend its quota of the £10,000 annually in providing scientific researches in its

own country; for it is only by the earnest work in the sick room, in the hospital ward, and in the pathological and chemical laboratory of many men in all countries that any scientific solution of these great and at present mysterious problems will ever be arrived at. The careful application of the numerical method will aid materially in this solution; hence, I hope that I have not occupied your time and attention altogether unprofitably in this brief review of the proceedings of the Public Health Section of the Ninth International Statistical Congress.

It is difficult, in my humble judgment, to exaggerate the importance of a properly regulated system of medical statistics in the study of disease, and I consider it to be extremely desirable that the question should be taken up seriously by the various medical societies in Great Britain.

The Statistical Society, under the guidance of those able and eminent statists, Drs. Farr and Guy, have set the example by awarding the Howard Medal of the past year to the best essay on the subject of Hospital Statistics. This was gained by Dr. Steele of Guy's Hospital, whose paper will be published in the next number of the *Journal of the Statistical Society*. I am no longer Editor of that Journal, and have not yet read the essay in question; but we have been informed by those who examined it on behalf of the Society (Drs. Guy and Farr, most competent authorities), that it is a very valuable contribution to our knowledge of the subject. Other essays of great merit on the same theme were also submitted, and one of them, by an able and experienced observer, will, I have reason to believe, be shortly published in a separate and expanded form. Among other things, it will show how very defective and injurious many of our hospitals must be in construction and arrangements, and how necessary an extended and searching inquiry into the whole subject is. Indeed, I believe that nothing short of a Royal Commission will be competent to deal with it in the manner required. If cattle plague and its injurious effects on our food supplies are of sufficient importance to demand the direct intervention of the State, are the preservation of human life from the unavoidable incidence of many of the accidents, injuries, and diseases to which it is liable, of less consequence to the welfare of the community, and more especially of that portion of the community which is unable to help itself, and is compelled to resort to public hospitals in the hour of distress and disease?

Our public charities are among the proudest and most creditable monuments of the benevolence of the nation, and

I am satisfied that by none would such an inquiry be more cordially welcomed than by those entrusted with their management. Since the late Sir James Simpson and Mr. Erichsen wrote upon the subject of hospitalism, a large amount of very valuable information has been collected which requires to be subjected to the patient investigation of a thoroughly competent and impartial tribunal; as, if it can be shown that inexpensive huts afford the best chance of restoration to health and of life to those struck down by accident or disease, and that costly palaces are liable to become abodes of pestilence, however well administered, some rules for our guidance in the future, so as to profit by the experience of the past, will doubtless be adopted in the interests of all concerned.

To statistical evidence alone will this consummation, devoutly to be desired, be chiefly due, and by it alone will it be possible, in the words of Professor Radicke of Bonn, "to stem the stream of baseless, and, to a great extent, erroneous doctrines by which medical science threatens to be overwhelmed."

JAMES FURNESS MARSON, F.R.C.S.

By the death of Mr. James Furness Marson, F.R.C.S., on the 17th of November 1877, the Society lost one of its oldest and most honoured members. Mr. Marson had been long a member of the Council, and more than once the Presidency of the Society was offered to him, but each time he had felt it necessary to decline the offer, in consequence of a painful malady to which he had become liable, and which, in the end, was fatal to him.

Mr. Marson was of very retiring habits, and was little known beyond the circle of his immediate friends. For forty years he held the post of Resident Medical Officer to the Small-pox and Vaccination Hospital; for as long a period he was the principal Vaccinator of the National Vaccination Establishment; and, from the time of the institution of Educational Vaccinating Stations, he was in charge of the chief station at Blackfriars Road, London. In the pursuit of these several duties, he obtained that consummate knowledge of small-pox and vaccination for which he became so widely known, and with which his name will be permanently associated.

Mr. Marson's principal published work was his elaborate paper in the *Transactions*, for 1853, of the Royal Medical and Chirurgical Society of London, entitled, "An Analytical Examination of all the cases admitted, during sixteen years, at the Small-pox and Vaccination Hospital, London, with a view to illustrate the Pathology of Small-pox and the Protective Influence of Vaccination, in degrees varying according as the Vaccination has been perfectly or imperfectly performed." At the time of his last illness Mr. Marson was employed in the preparation of a paper relating to the same subject, and embodying a further examination of the cases admitted into the Small-pox and Vaccination Hospital, for another sixteen years, if not for a longer period, subsequent to that included in his first paper. This paper was not completed at the time of his death, but it is understood that it was in such a state of preparedness as probably to admit of completion by certain of his friends.

ON PLAGUE AND TYPHUS FEVER IN INDIA.

By the President, Surgeon-General JOHN MURRAY, M.D.

(*Read: April 10th*, 1878.)

Pestilence has existed in the world from the earliest ages, and has caused great loss of life and extreme misery. There is great difficulty in diagnosing the diseases which have proved thus fatal, from the vague manner in which the symptoms are described. This obscurity is not diminished in the present day by some medical authorities questioning the identity of the plagues which have raged extensively of late years. Such visitations have been popularly attributed to Divine anger. We have many instances of this in the Jewish history, and in Homer we read of the displeasure of Apollo being manifested in the same manner. It is curious to find that the origin of the plague in Kumaon was attributed by the natives to a punishment inflicted by Devi on the high priest at Kiddernath.

The first notice of the appearance of the plague in India dates from the beginning of the present century, when there were cases of typhus, with occasional buboes. In 1816-17 plague raged in Kattywar, and continued to prevail till 1821, when it was seen by Dr. O'Shaughnessy, who is now in London, and has supplied me with useful information on the subject.

There is no record of its having extended to the interior of India till 1836, when it appeared at Palee, in Rajpootana. There were the ordinary typhus bubonic symptoms,* and it was very fatal. It spread to the surrounding villages in the cold season, and ceased on the advance of the hot season of 1837, but re-appeared in the cold season of 1837-8, of which attack a minute account is given in the second volume of

* Dr. Forbes says in No. II, p. 14,—"The buboes, in the mildest form, make their appearance with little constitutional disturbance, attended only by langour, debility, and general feeling of indisposition. In the most common variety, the occurrence of febrile symptoms, and the pain and swelling of the glands appear to be, in most cases, simultaneous, in many cases the buboes showed themselves before the fever, while in none were they developed at a later period than the second day of the disease. In the more violent and malignant form, no glandular swellings appear, or they remain small, hard, and exquisitely painful."

the *Transactions* of the Medical and Physical Society of Bombay.

It has not since appeared in Rajpootana with buboes. In the year 1837, a very fatal contagious fever, with similar symptoms, but without buboes, and with many of the symptoms of yellow fever,* raged in Rajpootana. This contagious fever spread northward, by Jeypore, to the Doab, and extended to Rohilcund, at the base of the Himalayas, where Kumaon and Gurhwal are situated. In 1836 information was received of several cases of plague† having appeared there, and inquiries were instituted on the subject. It was found that the disease had existed for 14 years in these districts, having first appeared in 1823 at the Temple of Kiddernath, in Gurhwal. This is one of the places of pilgrimage, at the edge of the perpetual snows, near one of the sources of the Ganges; and the tradition to which I have already alluded is, that the Rawal, or head priest, having deviated in some manner from the rites prescribed for the performance of the ceremony of the "Hom", died by a visitation of the anger of Devi, and that the other Brahmins who assisted at the offering were smitten and died also. The disease, thence arisen, spread to the villages or religious assignments connected with the temple, and afterwards extended to the villages in the route of pilgrimage, and proved fatal in 3,600 cases. It came lower and lower every year.

* Vomiting of bilious matter, and latterly of dark, coffee-coloured fluid comes on; the bowels are either constipated or the stools black and fœtid. In the most fatal form no buboes appear; anxiety and oppression of the chest, slight cough and bloody expectoration, clammy sweats, faintness and complete exhaustion, and a fatal syncope puts an end to the sufferings of the patient, generally within forty-eight hours from the attack. The intellectual faculties remain perfect till nearly the last moment.

† The symptoms of the disease in Kumaon, as described by Dr. Plank, p. 88 of his report, are considerably modified, especially as to the date of the appearance of the buboes. He says,—"Usually the first symptom of actual disease is shivering, followed by intense fever. After twelve hours, pain in the head will have become a prominent symptom; by the evening of the second day the sufferer will be delirious, by the evening of the third day the patient will be insensible, and die during the night, when no characteristic appearance of plague disease will be found in the body after death. In some cases the vital principle is overpowered at once by the violence of the disease, and the patient will die within twenty-four hours of the first attack; in one case, in twelve hours the man was almost insensible, pulse 150, eyes yellowish, not able to understand a question; a few hours after he died. In other cases, after the delirium or insensibility had continued for some hours, glandular enlargements appeared either in the groin, armpit, or neck. These may appear as early as the fourth day, or they may not be prominent till the seventeenth. The appearance of these glandular enlargements marks a favourable crisis in the disease."

There was a severe attack in 1834-5, and again in 1836-7 it was very fatal. In 1847 it appeared in a village 15 miles west of Almora, and the *Report* of Superintending Surgeon Renny, in 1850, describes its course in Kumaon during the previous year, though he did not consider it true plague. The native name under which it was alluded to in Kumaon and Palee is "Maha-murree", which signifies "great death", and applies equally to cholera, called by the natives "Kala-murree", or "black death". The local name for the plague is "Gola", a ball, or bubo. The nature of the disease was investigated by Dr. Pearson in 1851, and by him again, in conjunction with Dr. Francis, in 1852-3, and subsequently by Dr. Watson. It was considered by these officers to be the true bubonic plague, though it, as well as the Palee plague in Rajpootana, in 1836-7, had been supposed by some to be typhus fever, and not plague. The opinions of Drs. Pearson, Francis, and Watson have been fully confirmed by later experience, and the careful observations of Dr. Plank on the severe attack in Kumaon in 1876-7 leave no doubt of the existence of the characteristic symptoms of bubonic plague, with all the varieties common to the course of that disease. After the introduction of some sanitary measures in 1852-3, there was a diminution of the prevalence of the disease, but it appeared again in 1860 and in 1865, and in 1870, when a severe attack broke out at Sarkole. A very valuable and interesting report of the progress of the disease since that date has now been produced by Dr. Plank, the Sanitary Commissioner of the North West Provinces, in which the progress and predisposing causes of the disease are very clearly indicated.

I was at Meerut during the attack of contagious fever, previously mentioned, which visited the Doab in 1837.* No buboes occurred in the cases of the disease which came under my observation. In a few cases the parotid and glands of the neck suppurated, but I saw no enlargement of the glands of the groin or axilla. There was low fever, enlargement of the liver and spleen, yellowness of the skin and eyes, and black vomiting and purging, with a liquid state of the blood. These symptoms of yellow fever are found during all attacks of plague in individual cases, in

* The first case of this fever that was admitted under my charge at Meerut was nursed by his son, who was attacked when his father convalesced; he in his turn was attended by a friend, who, in the same manner, was attacked when he convalesced, and the friend died. Two native doctors were attacked, one of whom died, and nearly every one of the hospital attendants were attacked. The disease appeared to me to be an extension of the Palee Plague of 1836.

Palee, Kumaon, and other parts of the world, more especially in Eusufzaye, where a similar fever appeared in the end of 1852 and 1853. It was reported on by Drs. Farquhar and Lyell. Out of a population of 53,400, the deaths amounted to 8,433. At the commencement of the fever the face was flushed, with heavy, dull expression; the eyes were suffused, red, and in most cases the conjunctiva was yellow. A jaundiced condition of the skin was seen in by far the greater number of the cases, and indeed was a principal and early feature of the disease. The patient lay, usually, in a peculiarly apathetic manner; a few hours before death wandering was noticed, quickly followed by coma, insensibility, and death.

In severe cases death occurred on the second or third day, but usually the fifth or seventh was the fatal day. In less severe cases there was a remission from the eighth to the fourteenth day, and again often a remission on the twenty-eighth day. Inflammation and suppuration of the glands in the groin, axilla, and neck occurred in some cases that survived the first or second relapse. Some of these also were attended with sloughing, but this was not common.

The appearance of buboes is not an essential characteristic of plague. They are not present in the most intense cases. I consider them as merely a local symptom of a general disease, the appearance of which is very much influenced by season and temperature. It was observed in the Palee attack that they did not appear generally before the third or fourth day, whilst the most severe cases proved fatal before that date. This is more distinctly marked in the last attack in Kumaon, when Dr. Plank observed that the buboes may appear on the fourth day, or may not appear till the seventeenth. The majority of cases proved fatal on the third day, when no characteristic appearance of plague disease can be found in the body after death.

The close resemblance of the general action on the system of the poisons of plague, typhus, and yellow fevers, renders it probable that the local symptoms which supervene may be caused, as well as influenced, by local circumstances of season, habits, and surroundings. I see no distinct line of demarcation between plague, typhus and yellow fevers. Bubo is no characteristic of plague when all the worst cases prove fatal before it appears. In the Kumaon plague, of which 95 per cent. proved fatal often in two or three days, buboes were only found from the fourth to the seventeenth day. The jaundice of yellow fever is frequently found in the plague of Egypt, with bilious vomiting, followed by black

vomiting and purging. This was common with the plague at Palee, and it was characteristic of the extensive epidemic in the Doab, in 1837, where buboes did not appear in the groin or axilla, but the parotid glands suppurated in some instances. A disease similar to the epidemic of 1837 in the Doab and Rohilcund appeared at Sangor in 1859, and spread to Agra in 1860, and in the Central Jail there proved fatal in 313 cases. It was an intensely dangerous typhus fever, frequently accompanied with bilious vomiting, followed by black vomiting and purging, and jaundice, and very contagious. This disease has proved very fatal during the cold seasons from that date in several of the central prisons of the North West Provinces and the Punjaub, where it still lingers, probably on account of the climate being colder than in the North West Provinces.

The general character of the diseases we are considering, their communicable nature, the influence of season upon them, and the action of remedies, with in most instances their fatal course, classify them for practical purposes in the same category, if they are not caused by an identical poison. The symptoms of mental and bodily depression, followed by low typhus fever, are similar to the remittent fever caused by sleeping on low marshy ground at the end of the rainy season, particularly in the low land skirting the Himalayas, called the Terai, and in wooded marshy districts all over India. All the symptoms except buboes are found in the Terai fever, but it differs in not being transmissible from the sick, and also in the season at which it prevails. In all these diseases, the immediate direct depressing action of the poison is apparent, while the symptoms which supervene when the poison is not immediately fatal show the efforts made by nature to eliminate the poison from the system. The diagnosis between the typhus fever from the jails and remittent jungle fever cannot be made in many individual cases, though experience shows that the diseases have a different origin, and their course is different. The simultaneous appearance during the same epidemic of cases showing characteristic symptoms of what are considered specific diseases, may be explained by the different diseases being epidemic at the same time, and there are unquestionable examples of simultaneous attacks of distinct diseases. Typhus with small-pox or measles, and ague with plague or cholera.

In the present day some are found who attribute epidemic diseases to spontaneous origin from sanitary defects. These defects may unquestionably produce disease, which

proves fatal in a few hours, or is followed by typhus symptoms. An instance of this appeared in the fatal tragedy of the Black-Hole in Calcutta, when those who came out alive died afterwards of typhus fever. The same symptoms follow partial drowning, or strangling, or exposure to an atmosphere filled with carbonic acid, as the choke damp of mines, or fermenting vats; but, in these cases, the disease has not the power of reproduction. It is universally allowed that when once these contagious fevers appear, they may be disseminated by human agency, and though it is difficult in many instances to trace the connecting links, in my opinion defective information is more probable than the spontaneous origin of such specific diseases as plague or cholera. The rapidly fatal course of both these diseases is incompatible with a general or common origin either atmospheric or electric. Men apparently in robust health, surrounded by others in the same physical condition, are suddenly struck down and die in two or three hours, while their neighbours escape the slightest sign of disease. The transmission of plague by human intercourse has been clearly proved in Europe, and is an acknowledged fact. Indisputable cases of this transmission are recorded in the reports of Drs. Pearson and Plank on their recent investigations relative to the plague in Kumaon. We may probably trace the outbreak of plague at Kiddernath in 1823 to human agency instead of the vengeance of the Deity on account of maimed rites, as popularly believed. Plague we have seen existed in Kattywar in 1821, and most likely in subsequent years, though there is no official record of the fact. There are several sacred shrines in that district, and it is in accordance with Hindoo life for pilgrims to go from one to another. A pilgrim from Kattywar proceeding to Kiddernath may have conveyed the infection in his clothes or person, and thus communicated the disease to the Head Priest and his assistant Brahmins. These pilgrimages are one of the most fertile sources of the dissemination of cholera in India. The communicability of the disease was doubted in Kattywar in 1821; but it is firmly believed by the natives of Kumaon, as they show by their practice of deserting their villages and moving into the jungles when a case of gola appears. It is only small villages of one or two dozen houses that can be thus vacated. In the larger ones only the inhabitants of the infected houses move into the jungles. They desert their encampment for a new one should a fresh case appear in it. This practice has the double advantage of removing from the persons affected,

and also from the locality which becomes infected, and capable of communicating the disease. Instances were found in the recent investigations in which the disease reappeared on the return of the inhabitants to their houses. One very probable cause of the persistence of the disease in the districts of the Himalayas exists in the insanitary state of their dwellings and habits. Their elevation frees them from the miasmatic air of the plains, bnt their homes are small and ill ventilated, and their cattle occupy the lower story of the building. Their personal habits are filthy in the extreme; the saying is, that they wash their bodies once a year—in the rainy season—and change their clothes when they fall off in rags. The dissemination of plague and fever is promoted by the crowding of numbers in these small and ill-ventilated abodes in which the air is so foul. Dr. Watson considers that at night, and particularly during sleep, these deleterious agencies are most active, and in this I fully concur. The influence of the sun (or daylight) is a well known protection from the action of malarious poison in the Terai and other marshy and wooded localities in India. The susceptibility of the body to unhealthy influences during sleep and darkness is one of the objections to treating a large number of sick in one room, even though the cubic space and ventilation be satisfactory. Efforts are being made to improve the sanitary state of the villages in Gurhwal, and some progress has been made; we may, therefore, hope that this disease will disappear.

None of the medical officers engaged in investigating the outbreaks of plague in Kattywar, Palee, Kumaon, and Eusufzaye, were attacked by the disease. This may partly be attributed to their living in tents, apart from the villagers affected. In the attacks of the analogous fever which have taken place in the North West Provinces and Punjaub since 1860 in 68 jails, and where 5,346 cases proved fatal, only one civil surgeon was attacked, and he recovered. The native doctors and hospital attendants, more especially the sweepers, suffered severely in many instances.

In reference to the symptoms induced by the most intense action of the poisons of plague and cholera, viz., total prostration of nervous and muscular energy, I may mention that I saw similar symptoms induced in animals by the action of snake poison, introduced by the bite of a snake or the transfusion of the poison into the veins of dogs, during some experiments made in Calcutta by Sir Joseph Fayrer, the difference being that the extreme prostration, without active symptoms, terminated in death in a few minutes—from ten

to twenty. All remedial measures were powerless, and the rapid painless extinction of life was appalling.

Dr. Forbes mentions in his report (p. 258) that cases of poisonous snake bite are of constant occurrence in Marwar. In those where recovery takes place the blood becomes dark and liquid, low typhus symptoms supervene, with bloody vomiting and purging, and yellowness of the eyes and skin. In less severe cases, the bloody vomiting and purging do not appear, nor in slight cases does the yellowness supervene. These symptoms closely resemble those induced by the plague poison in some epidemics in Kumaon and Palee, and also in Persia, Egypt, and Europe.

These experiments were instituted for the purpose of obtaining an antidote to the snake poison, but they proved that the many supposed remedies were powerless. The same remark applies to the action of remedies in extreme cases of plague or cholera. In those less severe the most successful treatment is not directed to finding an antidote to the poison, but to supporting the strength and obviating complications, whilst Nature, the *vis medicatrix naturæ*, eliminates the poison from the system. Violent or strong remedies are not only powerless for good, but injurious, and none more so than excess of alcoholic stimuli. The natural functions should be promoted by mild remedies, and the strength supported by light nourishing diet. The spirits should be cheered, and the discomforts alleviated by good nursing; that this last is not a dangerous occupation, it is worse than folly to assert.

We can also save life by removing the insanitary causes which foster the poison, and we can obviate the danger of transmission by the isolation of the sick, and the removal of the healthy from the infected locality as far as practicable. These questions of the isolation of the sick and removal of the healthy from the infected locality are very summarily disposed of by the inhabitants of Gurhwal and Kumaon on the appearance of plague among them, for the village is deserted, and the sick are left to die alone. The guiding principle is sound, but their practice is cruel. Under proper regulation this principle has been applied in India with manifest advantage to the removal of troops and prisoners in outbreaks of cholera epidemics, and of typhus fever in the jails. The benefit of removing the prisoners into camp from the jails in typhus epidemics was not so great as in those of cholera, which may arise from the difference of the diseases, as *cholera may, whilst typhus cannot, be cut short* in its earliest stage, but must run its course. The benefit

of removal of the troops from cantonments in cholera epidemics is unquestionable. If the mortality from cholera amongst the European troops in India for the last thirteen years had continued at the same ratio as that of the thirteen years before removal was established, it would have been 5,340 instead of 1,994, which actually took place. I see in *The Times* of the 3rd April that the mortality from the epidemic typhus disease now prevailing among the Russian troops in Bulgaria amounts to 10 in the 1,000 per diem. Such appalling mortality from an unseen enemy is apt to demoralise the troops, and to shake the nerves of the medical officers, who find their remedies powerless to save their patients. Removal might well be tried in such a case, though it may be said it is not applicable to troops on service, as they are already in tents; but that would be overlooking the fact that it is in fixed camps, during sieges or after active service, that this disease rages most severely, and that in these the conservancy arrangements are often very imperfect, therefore removal to fresh ground would be as advantageous from them as from cantonments.

In the Kumaon Reports on plague instances are given of the disease appearing in a milder form, though still of a very fatal character. The natives called this type of the disease "Sanjar". Its symptoms and communicable nature are similar to the Maha-murree "Gola", but without the buboes. This disease appears very similar to the camp fever which is proving so fatal in Armenia and Bulgaria.

The influence of season is very clearly defined, and shown to be independent of temperature. The disease prevailed in Kumaon most severely during the cold season, from November till May, and subsided during the hot season and rains. This coincides with the course of the typhus fevers which have proved so fatal in the jails in the North West Provinces and the Punjaub since 1860. We find, from the accounts of the plague in former days, in Egypt, Syria, and Constantinople, that it prevailed in the cold season, and subsided as the heats of summer advanced. Plague has been prevailing for the last three years in Persia and in the valley of the Euphrates, and it is similarly influenced by season.

The accompanying Table shows the seasonal mortality in forty villages inspected by Dr. Plank, in 1876-7, compared with a similar return of the plague in the valley of the Euphrates and in Persia for the same years. The period is too short to obviate the possibility of these years having been exceptional, but the result is consistent with general views derived from other sources.

Table showing the influence of Season on Plague in Kumaon and Mesopotamia in 1876-77, *and Typhoid Fever in the Upper Provinces of India, and on Cholera in the Upper Provinces from* 1817 *to* 1877, *and Miasmatic Fever from* 1864 *to* 1875.

			Jan.	Feb.	March.	April.	May.	June.	July.	August.	Sept.	Oct.	Nov.	Dec.	Total.
I.	Plague in Kumaon	Deaths	53	11	38	54	14	5	3	3	23	23	25	58	277
		Ratio to Deaths	19·13	3·79	13·71	19·49	5·05	1·80	...	1·08	1·08	8·34	9·02	20·93	
II.	Plague in Bagdad	Deaths	58	102	350	2,994	2,590	176	7	...	...	...	...	...	6,318
		Ratio to Deaths	0·92	1·62	5·57	47·66	39·94	2·77	0·11	...	...	...	...	...	
III.	Typhoid, Up. Prov.	Deaths	516	589	864	876	482	189	103	118	258	389	422	490	5,346
		Ratio to Deaths	9·65	11·02	16·16	16·35	9·01	3·53	1·92	2·77	4·82	7·22	7·89	9·16	5,346
IV.	Cholera, Up. Prov.	Deaths	6	11	76	209	436	604	1,125	3,005	1,435	261	192	25	7,403
		Ratio to Deaths	0·08	0·12	1·02	2·82	5·88	8·19	15·19	40·56	19·61	3·52	2·59	0·34	
V.	Ague, Punjaub	Cases	6,022	4,079	3,949	4,318	5,399	6,384	6,490	11,456	21,952	30,342	23,582	10·611	135,614
		Ratio to Cases	4·44	3·00	2·91	3·18	3·98	4·50	4·79	8·44	16·18	22·37	17·39	7·82	
VI.	Ague, Bengal	Cases	3,782	2,850	3,460	5,312	6,838	5,746	6,385	7,165	6,266	6,502	6,724	5·106	66,333
		Ratio to Cases	5·70	4·29	5·21	8·01	10·35	8·66	9·62	10·80	9·44	9·80	10·13	7·66	

I.—Plague in the civil population of Kumaon, 1876–77.
II.—Plague in the civil population of Bagdad and Stella, 1876-77.
III.—Typhoid yellow fever among the native prisoners in the N.W. Provinces and Punjaub from 1859 to 1875.
IV.—Epidemic cholera among the European troops in the N.W. Provinces and Punjaub from 1817 to 1875.
V.—Endemic miasmatic fever among the native troops in the Punjaub from 1864 to 1875.
VI.—Endemic miasmatic fever among the native troops in Bengal from 1864 to 1875.

It also shows the seasonal coincidence of the plague in Kumaon, and of the typhus fever in the jails in the North West Provinces and the Punjaub from 1859 to 1875, and the different seasons in which epidemic cholera and endemic fevers prevail in the same parts of India.

ON THE CHARACTERS OF EPIDEMIC PLAGUE IN MESOPOTAMIA IN 1876-77.

By E. D. DICKSON, M.D.,
Physician to the British Embassy, Constantinople.

(*Read*: *February 26th*, 1879.)

The sudden and alarming announcement of an outbreak of plague in Asiatic Russia; the possibility of this scourge extending into Europe; the conflicting opinions that seem to be entertained concerning the nature of the malady and the precautions necessary to ward it off, have prompted the following brief sketch, derived almost entirely from notes taken, and kindly given me, by Dr. Giovanni Cabiadis, as the result of his observations at Hillah and Bagdad, during the outbreaks of plague in 1876-77.

History.—Two or three months previous to the manifestation of plague in Mesopotamia *glandular swellings free from fever* prevailed in that country. They showed themselves in the groin, armpit, or neck, and were not accompanied by other symptoms. They began to appear amongst the inhabitants about the end of autumn, and continued through the winter; but towards the end of it plague broke out and reached its acme of intensity in the spring, and died out suddenly during the summer season when the *great heat* declared itself, and the thermometer rose to 45 or 50 degrees of centigrade scale (113 degs. or 122 degs. F.). During the prevalence of the plague the thermometer ranged between 5 and 30 degrees; and when it rose to 30 degs. (86 degs. F.) the disease had reached its maximum of intensity. As the temperature increased from 30 degs. to 45 degs. (86 degs. to 113 degs. F.) the epidemic began to diminish, and as soon as the thermometer got up to 45 degs. (113 F.) it ceased abruptly. On the cessation of plague *apyretic glandular swellings* reappeared again, precisely similar to those which had preceded the outbreak, and they continued to manifest themselves for about two months longer. These glandular swellings were frequently met with, and were distinct from the chronic adenitic swel-

lings met with in subjects of a scrofulous tendency, and evidently unconnected with any special diathesis.

Symptoms.—The symptoms that characterise plague are of two orders, local and general. In severe cases the general symptoms precede the local manifestations. The patient is suddenly seized with high fever, which, in some instances, is ushered in by a prolonged shivering fit, the pulse beats 130, or even more, and the temperature of the body rises to above 42 degs. (107.6 degs. F.) centigrade. In some cases, however, this febrile condition is hardly appreciable, while on the other hand alarming complications indicate the severity of the attack by perturbations connected with the nervous centres, such as convulsive tremor, drowsiness, coma, delirium; or with the circulating system, such as epistaxis, hæmatemesis, hæmoptysis, sanguineous diarrhœa, menorrhagia; or with the assimilative organs, such as painful tumefaction of the liver, bilious vomiting, bilious diarrhœa, jaundice, etc. These phenomena appeared to be dependent on some *periodic impulse,* because at times the bilio-gastric manifestations prevailed; at others the hæmorrhagic, and at others the nervous. The nervous agitation which sometimes ushers in an attack of plague is a very remarkable symptom, and one that Dr. Cabiadis had never met with before. He describes it as a *prolonged regular shake* lasting from six hours to three days. While the patient is suffering from it he does not complain of cold, nor does the thermometer indicate a lowered temperature. The pulse is small, short, and quick, and the temperature of the body remains in nearly its normal state. This tremor is always followed by profound coma, during which the patient rapidly sinks.

In some instances of plague Dr. Cabiadis has seen death supervene within a few hours of the attack, and before any of the characteristic indications of the disease, such as buboes and carbuncles, had shown themselves. These severe cases were mostly met with at the *commencement* and at the *termination* of an epidemic outbreak.* They were observed in the plague of 1876 at Hillah, as well as in that of 1877 at Bagdad; and in these instances the glandular swellings either did not appear at all, or were very small and never suppurated; the patient, moreover, seldom lived more than three days. The cutaneous surface, in some cases, was so filled with *petechiæ* that, when death super-

* I have noted a similar circumstance in severe epidemic outbreaks of *cholera.* The first and the last attacks were mostly *fatal.*

vened the skin assumed a dark livid hue, giving the corpse a *blackened appearance*, and so characteristic of the malady that it might even to this day be called the *black death*.

In the usual course of plague, however, the local manifestations precede the general symptoms. The patient first complains of pain in the groin, the armpit, or the neck, and on examining the part a swelling is found there, which rapidly increases and usually suppurates by the seventh or eighth day; at the same time carbuncles may show themselves, and in bad cases petechiæ. In these instances the fever runs high and in accordance with the gravity of the case. When *death* supervenes it mostly occurs on the fourth or fifth day of the illness; but three-fourths of such patients are reckoned by Dr. Cabiadis to get well.

In slight attacks of plague the local manifestations are seldom accompanied with *fever*, and you often meet such patients walking about the streets, with two or three suppurating buboes upon them. These cases are hardly ever fatal.

I have expressly omitted, in the foregoing description, to mention several symptoms which are usually met with in all febrile complaints, in order to draw attention to those which may be considered as pathognomonic of the plague. Headache, thirst, constipated bowels, lassitude, and aches in the limbs exist, more or less, in all fevers. The appearance of the tongue in cases of plague is not in general much altered; usually, it is red at the tip and edges, and sometimes dry. A comatose state is much more frequently met with than a delirious one. The diarrhœa is always either sanguineous or bilious, but never *serous*. As a rule an attack of plague lasts from a few hours to four weeks, and Dr. Cabiadis considers that one-third of the attacks end fatally. From the accompanying table, however, of 1,826 cases of plague seen and registered by him at Hillah in 1876, the proportion of deaths to that of the attacks is 52.6 per cent. But a great many cases of plague, he observes, are never made known, and would thus diminish very considerably this per-centage of its mortality, whilst the deaths, on the other hand, must all be reported, in order to obtain the permit of interment.*

* The same remark applies to the death-rate shown by the official register kept at Bagdad in 1876, and which gives 55.7 as the percentage of deaths to that of the attacks.

ANALYTICAL STATEMENT of 1,826 Cases of Plague noted by Dr. Cabiadis at Hillah, during the Epidemic of 1876.

Age.		Sex.		Result.	
From 2 months to 9 years	277				
„ 10 years „ 19 „	617				
„ 20 „ „ 29 „	432				
„ 30 „ „ 39 „	292	Males	889	Got well	865
„ 40 „ „ 49 „	123				
„ 50 „ „ 59 „	52				
„ 60 „ „ 69 „	18	Females	937	Died	961
„ 70 „ „ 79 „	11				
„ 80 „ „ 89 „	3				
An old man of 113 „	1				
Total	1,826		1,826		1,826

Manifestations:—

Glandular swellings—		
In the groin	710	
„ axilla	466	
„ neck	98	
„ several places	122	
	1,396	
Carbuncles	36	
Dependent on the nervous centres — Coma		28
Convulsive shake		9
„ circulating system — Petechiæ		120
Epistaxis		2
Hæmoptysis		6
Hæmatemesis		27
Sanguineous diarrhœa		14
Menorrhagia		2
„ assimilative organs — Bilious vomiting		32
Bilious diarrhœa		16
Jaundice		2

Diagnosis.—The diagnosis of plague is not difficult, for no other malady presents an assemblage of symptoms so well-marked and so significant of its true nature. In fact, no other idiopathic fever, attacking a multitude of persons at the same time, is characterized by glandular swellings, by carbuncles, and by those severe manifestations of the nervous, sanguineous, and bilious systems which declare themselves in an attack of the *plague*.

Dr. Beck, and a few other physicians, basing their views on the intercurrent phenomena manifested in some cases of plague, imagined that this disease might be merely a variety of the *pernicious fevers* engendered by the *malaria poison;* but Dr. Cabiadis cites three facts which conclusively subvert this hypothesis.

1. No *intermission* has ever been observed in the symp-

toms of plague, not even in those intercurrent phenomena which arise from complications of the nervous, circulating, and assimilative organs.

2. There is no instance on record of an attack of plague having been *cut short by the administration of sulphate of quinine.*

3. The striking difference which marks the *expression of the countenance* and the *general aspect* presented by a person suffering from an attack of *pernicious fever*, and those of one suffering from the *plague*. On coming up to a patient suffering from an attack of pernicious fever you are struck with the gravity of his case and the danger threatening his life. The very reverse of this meets your eye when you see, for the first time, a case of plague. Even the worst instances of this malady are apt to deceive an *inexperienced* physician, and make him fancy that the case is free from danger, when in reality the patient has only a few hours to live. The first instance of plague seen by Dr. Cabiadis did not seem to him to be one of an alarming nature. The patient looked stupefied, or rather as if intoxicated, and did not answer readily the questions put to him. He had vomited blood, and had a very small bubo in the right axilla, but the pulse and temperature were normal. This patient *died a few hours after Dr. Cabiadis' visit.*

Prognosis.—Rapid suppuration of the buboes, even when accompanied with *high fever*, indicates a favourable termination.

All cases complicated with nervous, hæmorrhagic, or bilious manifestations, end fatally.

Contagion.—With regard to the debated question of *contagion*, I beg to draw attention to the conclusions arrived at by Dr. Cabiadis, and which are derived from a careful study of the facts that came under his observation during the two last outbreaks of plague in Mesopotamia.

Dr. Cabiadis neither attempts to deny the contagious properties of plague nor does he accept unconditionally the assertion of those who pretend that you must keep at a distance, say of six metres, from the patient in order to avoid contracting the disease. The experience acquired by him during the epidemic outbreaks of Hillah and Bagdad has convinced him that no great risk is incurred *in touching persons affected with the plague*, provided you avoid exposing yourself for any lengthened period to the atmosphere of the apartment occupied by him. As a proof of this doctrine, he says that those persons who lived in the same house with a plague-smitten patient, but who avoided touching him or

his clothes, through fear of catching the infection, generally did get the plague; whilst those who lived in houses exempt from the malady, but who visited plague patients and handled them freely, without remaining long in the same room with them, hardly ever caught the disease. When plague entered a house it seldom spared its inmates, and often carried them all off within a week's time. In noting these facts, Dr. Cabiadis was careful to take into account the various tenets of the different religious sects of the people of Bagdad; for, while the Mohammedans, discarding all sanitary precautions, lived with and handled plague patients, the Jews and Christians, on the contrary, firmly believing in its contagious properties, very seldom *touched them;* yet all of them equally caught the disease if they *lived in the same house* with the plague patient, but were safe if they *quitted the house* the moment a case of plague occurred in it. Dr. Cabiadis quotes another remarkable fact in support of his theory. With *one exception,* none of the physicians, surgeons, or assistants who daily attended on plague patients *caught that malady,* and he himself handled nearly 2,000 plague-smitten persons without suffering any inconvenience from it. To this it might be objected that the immunity enjoyed by them was owing to their *contact with the sick being a momentary one.* But, Dr. Cabiadis remarks, that if this be true with respect to the *physician,* it cannot be so with regard to the surgeon and assistant, whose duties obliged them to prolong their stay near the patients, for the purpose of opening abscesses, washing and dressing sores, etc. The exception alluded to above was that of *an assistant who actually died of the plague.* But in an epidemic outbreak affecting a whole population, one death amongst an entire corporation would rather indicate the law that governed the general mortality at the time than the exceptional liability of that body for coming into contact with the diseased.

Dr. Cabiadis does not think that the *infectious matter* of the plague is contained in the *pus secreted from the buboes and sores.* Plague, he observes, is transmitted even in its *incipient stage,* and before any of the swellings or boils have suppurated. The problem therefore remains to be solved whether the *specific materies morbi* of plague is exhaled from the patient's respiratory organs, from his cutaneous surface, or from some other emunctory.

These considerations lead me to the important practical question of *fomites.* I was very anxious to obtain from Dr. Cabiadis the fullest information on this point, so as to clear

up if possible this intricate subject. Dr. Cabiadis says that he considered the *atmosphere* which surrounds a person affected with plague as *the true medium of transmission.* Apart from this, he has also met numerous instances in which he believes the *clothes and bedding* of plague patients communicated the malady to sound persons living beyond the sphere of the epidemic outbreak. Such articles were made of wool, cotton, flax, or hemp. He ignores whether these materials in a *raw state* would have given the disease as well, since he has not met with any fact to enlighten him upon the subject.

Etiology.—The origin of plague and the causes that bring it into activity are utterly unknown. Some persons have conceived the idea that a *marsh-miasma* might be the exciting cause of this pestilence, but Dr. Cabiadis refutes this idea by quoting the immunity from plague enjoyed by Bussorah and Kerbela—two of the dampest cities in Mesopotamia, and both of them *surrounded by marshes.* Kerbela, in fact, stands on an island in the midst of a marsh, the waters of which reach up to its very walls. Midhat Pasha, when Governor-General of Bagdad, built a fine new quarter at its south-west end. The whole of it, however, has now fallen down, in consequence of the foundations being embedded in a slushy soil. Kerbela, therefore, according to the malaria hypothesis, ought to be a spot favourable to the development of plague, and yet it has not been visited by that epidemic, notwithstanding that all the surrounding villages have been more or less attacked by it, and that persons suffering from the plague have gone to Kerbela and died there.

Some persons have fancied that eating *diseased camel's meat* might have caused the outbreak in Mesopotamia. In order to find out whether there were any truth in this assertion, Dr. Cabiadis made a careful inquiry, but failed to discover any evidence in corroboration of it. He therefore infers that diseased camel's meat, like that of any other diseased animal, might *predispose* to an attack of plague, but not engender the malady.

Proximate Cause.—The most palpable and evident of all the causes which predispose an individual to an attack of plague during an epidemic outbreak is *poverty.* No other malady shows the influence of this factor in so striking a degree; so much so, indeed, that Dr. Cabiadis styles the plague *miseriæ morbus.* In his experience, he found that the poor were seldom spared, the wealthy hardly ever attacked. *Cholera* also has a preference for the poor, but,

at the same time, it spares not the rich, whereas plague attacks exclusively the poor. Here, again, Kerbela presents a striking proof of this fact. Its inhabitants are the most prosperous section of the whole population of Irak-Arabi (Turkish Arabia), and even its poorest classes can at all times live on a meat diet. Its streets are narrow and crooked, but the houses are spacious and well aired. Hillah is the very reverse of this. Its houses are low, confined, and very imperfectly ventilated; they are, moreover, generally encumbered with a horse, with poultry, and with two or three buffaloes. These animals constitute the resources whence the lower classes of Hillah derive a livelihood by selling milk and eggs to the wealthier inhabitants, while they themselves limit their own nourishment to barley bread, dates, and onions, with sometimes fish in a putrescent state. It is needless to add that Hillah suffered severely from the plague.

Prophylactic Measures.—The most effectual means for the protection of a community against the propagation of plague—the isolation of the sick, the destruction by fire of their clothes, and the whitewashing with lime and free ventilating of the domiciles in which cases of plague occur. The plague *reappeared in only one* out of 350 houses which had been *whitewashed* after an outbreak of plague in them, whilst its reappearance in houses that had merely been abandoned for a time, after an attack of that disease, but which had not been whitewashed, *was of frequent occurrence.*

The other disinfectants tried, such as sulphur fumigations, lighting fires, and throwing a solution of sulphate of iron into the drains, gave no decided results, except when associated with the whitewashing and airing of the infected house.

Cordons Sanitaires.—Dr. Cabiadis believes that *cordons sanitaires* properly enforced are extremely useful in checking the extension of an outbreak of plague. The greatest vigilance, however, is required to render them efficient, because the persons put to guard these cordons often allow passengers to evade them, either through neglect of their duties, or through the persuasive influence of bribes. He strongly condemns, however, the practice adopted at one time in Bagdad of shutting up persons smitten with the plague in their houses, and placing guards round them to prevent communication with the rest of the population. The fear of this harsh and unreasonable measure caused the inhabitants to *conceal* the existence of the malady, and even *to bury inside the house* those who succumbed to it, thus

deceiving the authorities, and helping to intensify and propagate the evil it was intended to mitigate.

Treatment.—Dr. Cabiadis can offer no suggestions worthy of trust as to the best mode of treating plague. The remedies chiefly tried at Bagdad and at Hillah were leeches, and mercurial frictions to the swellings, combined with the internal administration of phenic acid, or sulphate of quinine. In some cases these remedies were thought to do good, while in others they proved useless, if not detrimental, and Dr. Cabiadis is convinced that we know as little about the cure of this disease as we do about its essence.

He concludes his interesting notes on this subject by expressing astonishment and regret that no public hospital exists in Bagdad, an extensive city of 150,000 inhabitants.* A large *military* hospital was built there by Midhat Pasha, on a fine and healthy site, but no use has been made of it. He therefore suggests that the Government should turn it into a *civil hospital,* for the benefit of the people of Bagdad.

* Population of Bagdad—

Shiah Mohammedans	70,000
Jews	30,000
Christians (chiefly Chaldeans) and Soonnee Mohammedans	50,000
Total	150,000

REMARKS ON THE DIAGNOSIS AND CAUSE OF YELLOW FEVER.

By ROBERT LAWSON,
Inspector-General of Hospitals.

(*Read: March 5th*, 1879.)

Though the literature of yellow fever be very extensive, and highly controversial, the epidemiological questions discussed in it are but few, and I shall occupy the limited time at my disposal to the greatest advantage, and meet the objects of the Society most effectually, by confining to these the remarks I am about to offer, and endeavouring to indicate where the truth lies between the contending parties. In what follows, particular manifestations of the disease will be referred to, to illustrate the mode of investigation and the conclusions arrived at, but these must not be considered as by any means exhausting the evidence that has been accumulated on the same points.

From the time of the earliest definite records of yellow fever to the present, there have been two opinions as to the nature of the disease, and the manner in which it was propagated. When the early colonists first came in contact with it, they naturally thought it allied to plague, the most deadly disease they were acquainted with, and which was then common in Europe, and it was not surprising that in their endeavours to account for its appearance and spread, they should have assumed it to have been imported in the first instance, and subsequently to have been propagated by contagion from man to man; but a very short experience led many observant people to doubt those positions; and we find Lygon, who arrived in Barbadoes in September 1647, and published his history of the island two years after, while he admitted it was doubtful whether the disease had been carried there in ships, or caused by the irregularities of the inhabitants, added that he had "reason to believe the latter; because, for one woman that died, there were ten men; and the men were the greater *deboystes*" (*i.e.*, the more debauched). Lygon also remarked on the

very unhealthy site of Bridgetown, the first settlement in Barbadoes, as being beside a small creek which he describes as quite a morass, which, indeed, it continued to be until a comparatively recent date. With every succeeding pandemic extension of yellow fever the same question has been raised; and it is worthy of remark, that while the great majority of those residing in the yellow fever localities in the West Indies, both lay and medical, regarded the disease as endemic, and as not communicable from the sick to those in health, the advocates of importation and contagion increased in numbers in places where the disease was only met with occasionally, as in the United States, and may be said to have attained their greatest strength in those parts of Europe where they had little or no experience of it. Nor does this diversity of opinion seem likely to cease; the latest work on yellow fever, by M. Bérenger Féraud, a medical officer of the French Marine, published since the commencement of this year, prophecies an early assent of the medical profession to his views; viz., that the disease is communicated from man to man; and the Commission in America, which has reported on the recent epidemic in the Valley of the Mississippi, has adopted certain opinions, and based on them recommendations as to prevention, which have already called forth considerable criticism, and which there seems reason to believe it will have some difficulty in establishing.

At a very early period of the controversy, supporters of the importation theory accused their opponents of confounding two distinct diseases, the ardent from the ordinary endemic, with the yellow fever, which they held to be of a truly pestilential kind, and highly contagious. This part of the controversy, also, has come down to us in principle, though the details have been gradually modified to meet the increasing acquaintance with the circumstances under which the disease manifests itself. A typical case of ardent or remittent fever can be distinguished readily from an equally well marked one of yellow fever; but as all these may occur at the same place, and even sometimes at the same season, and as there are always numerous cases in which the distinctive symptoms do not come out so prominently as in the typical ones, it was frequently found difficult to determine whether the disease in any particular individual were yellow fever or one of the other forms; this led many, who considered yellow fever to originate from local causes, to regard it as merely a more intense grade of the ordinary endemic fever, due to a more con-

centrated emanation, and appearing for the most part in unacclimatised Europeans. The addition to our knowledge of the characters of yellow fever during the last thirty years, shows clearly that it forms a distinct species, differing from both the ardent and the remittent, though like them it is met with in every degree of severity, from little more than an ordinary ephemeral attack, without danger, to the most intense and deadly fever. So far, therefore, those who advocated the influence of local causes were wrong, but this does not affect the other portion of their position, that the yellow fever was connected with locality, and not due to importation, in support of which they adduced not only frequent sporadic cases between the different epidemics, but even many considerable outbreaks for which no source of importation could be established. To get over this difficulty the supporters of the importation theory long ago described certain cases of the ordinary endemic fever, in which yellowness of surface, and a kind of vomit, closely resembling black vomit, were met with, which, they maintained, constituted the form of disease their opponents referred to as originating from local causes, and not what the former held to be the contagious yellow fever. This explanation was considered sufficient half a century ago, but since then it has been found necessary to be more specific, and the opinion is now set forth that there are two species of yellow fever, the malarial and the specific, which so closely resemble each other in their symptoms, including yellowness of surface and black vomit, that the former can be distinguished from the latter only by its not presenting albumen in the urine, and not extending from man to man. The former of these is said to be the disease which is due to local causes, and to which the sporadic cases are to be referred; and the practitioners in the West Indies are said to mistake this habitually for the specific disease, which, it is maintained, arises there from contagion only, and is but an occasional visitant.

It becomes necessary, therefore, to define what yellow fever really is; and here it must be remembered, that every symptom it presents is met with in other diseases, and one or more of the more prominent may even be absent in individual cases; no single symptom, therefore, can be considered as pathognomonic, but its distinctive characters depend on their grouping, and the succession in which they occur during its progress. The following are its characters:—

1. Yellow fever is a disease which usually terminates in

convalescence or death, from the fourth to the seventh day; but either may occur as early as the second, or not before the tenth or twelfth, or even later.

2. There is generally yellowness of the eyes and surface, commencing at various periods in different individuals and epidemics.

3. On the evening of the third day, or morning of the fourth, the urine usually presents traces of albumen; on the latter a considerable sediment appears in it, consisting almost wholly of scaly epithelium from the bladder; this is succeeded by an equally copious one on the morning of the fifth day, which consists almost exclusively of granular tube casts from the kidneys, with scarce a trace of epithelium from the bladder. By this time the albumen has usually become considerable, the chlorides and urea have been greatly reduced, and the urine, as a whole, is usually scanty, and may even go on to complete suppression; if there be much yellowness, it may contain a variable quantity of the colouring matter of bile.

4. The alvine discharges are devoid of the natural feculent appearance from the third day onwards, becoming greyish or yellowish white, with quantities of black matter diffused through it, when they are formed; when fluid, consisting of mucus tinged with the preceding, or with bile, or blood, in variable quantities. On the approach of convalescence, the evacuations resume their natural colours.

5. As the urinary and alvine evacuations assume these characters there is a great tendency to black vomit, or discharges of similar matter from the bowels, or to the so-called hæmorrhages from the various mucous surfaces, or even, in some cases, from the skin, and, after death, such may be found in the stomach or intestines, when they had not been discharged during life.

Such are the distinctive features of a normal case of yellow fever; but in some the urinary symptoms may occur earlier than here mentioned, and in others they seem delayed for a day or two, but whenever watched from day to day, and properly examined, it is found that the changes in the urine not only embrace the presence of albumen, but indicate desquamation of the bladder and kidneys as regular features of the disease, and when these are fully developed the urea is much diminished, and the chlorides almost, if if not completely absent. It is important to bear these facts in mind, for albumen has been found in the urine during the cold stage in certain intermittents and remittents, but in such it went off as the fever came on, and

was not preceded or accompanied by desquamation of the bladder and kidneys, and loss of chlorides, characteristic of yellow fever, but which are not met with in pure intermittent or remittent. After death from yellow fever, the microscope shows acute exudation into the parenchyma of the liver, and when there has been yellowness, the minute bile ducts are found to be occluded; the kidneys also show acute exudation around the convoluted tubes, and the lumen of the latter often altogether obliterated by granular epithelium; and, as a constant feature, there is also acute exudation into the muscular substance of the heart, the fibres of which lose their striæ more or less completely, and show a great disposition to split up into their component fibrillæ, while the voluntary muscles in the same subjects present their natural appearance. The affection of the liver may be met with in cases of the ordinary form attended with yellowness, but those of the kidneys and heart are confined to yellow fever.

There has long been a contention between the two parties as to the form of yellow fever, the Contagionists affirming that it is always continued with a paroxysm of about seventy-two hours, or a little more or less, followed by the characteristic symptoms of the disease, while their opponents maintain that the latter are often found as fully developed in cases in which the fever has been distinctly remittent, or even intermittent, as in those which present the single paroxysm. Previous to our becoming acquainted with the urinary symptoms, when the line of demarcation between the ordinary forms of pure and yellow fever could not be drawn with precision, there might have been sufficient uncertainty on this point to afford ground for ardent controversialists to differ upon, but the trustworthy diagnosis made possible by the present knowledge of these has enabled numerous observers, in different countries, to verify the fact that yellow fever is met with not only in the continued form, but frequently as a remittent and, from time to time, even as an intermittent. Whether the continued form be that proper to the disease, and the other hybrid forms impressed on it when the ordinary malaria, which gives rise to periodic form, is rife, need not be enquired into at this time, it is sufficient for the present purpose that it be clearly understood that a fever, presenting the characters set forth above as those of yellow fever, occurs both sporadically and epidemically in a periodic, as well as a continued form.

The doubts as to the nature of the disease having thus

been removed, the circumstances under which it appears may next be examined. The point for consideration here is whether the disease be connected with a locality more or less circumscribed, in which the cause is generated independently of persons already labouring under it, or whether it depend on a morbid poison given off by the sick, which developes the disease among susceptible persons exposed to it, in whom the poison is augmented, and each of whom may so form a fresh centre for its extension. Before approaching this question, it is necessary to show the nature and bearing of the evidence by which alone it can be decided. There is no test known for the presumed activity of the yellow fever poison but the reaction of the living bodies of men, or of animals that are subject to the disease, and when cases of it arise in any locality where it is a frequent or even only an occasional visitant, all that that proves is that its causes are in operation there. If the persons affected have not been away from the locality for a considerable period, and there be no evidence of its having been introduced from elsewhere, the inference must naturally be that the disease arose from factors in operation at the place. If, however, one or more individuals labouring under yellow fever had arrived from another locality shortly before; and if the first person attacked had been in communication with the sick, whether immediately or mediately, it might be supposed that a poison was conveyed from the latter to which the seizure was attributable; there would be no means, however, in such an instance, of arriving at a conclusion either way, for, to establish the communication between the imported cases and the healthy person, the possibility of local causes having acted must first be excluded; and as the occurrence of the disease in the new locality merely shows the exciting cause to be in action there at the time, there is nothing to indicate whether that was connected with the imported cases, or was quite independent of them. The great majority of the examples adduced in support of the contagiousness of yellow fever are of this description, and when legitimately interpreted, the evidence they afford is quite insufficient to establish the conclusion drawn from it, provided the *de novo* origin is proved to be possible.

There are numerous well-authenticated facts, however, which show that, when yellow fever has sprung up in a locality, persons going to that locality may contract the disease, and should they leave before it become developed and go to a healthy one, they pass through the disease

without affecting anyone about them, and this even when their numbers are considerable. I alluded to a striking instance of this in a paper read before the Society in 1874,* the salient points of which may be repeated here. There was an epidemic of yellow fever at Sierra Leone in 1865, and the *Isis*, receiving ship there, became affected at the end of the year. The *Bristol* frigate arrived from England late in December, and anchored four or five miles to seaward of the usual place where the *Isis* lay. It being considered necessary to alter the berth of the latter, a party of 116 officers and men were sent from the *Bristol* to her on 28th and 29th Dec. for this purpose; these men left the *Bristol* in the morning, and returned to her each night, without communicating with the shore. Two of them were attacked with yellow fever on Dec. 31st, twenty on January 1st, six on 2nd, three on 3rd, three on 4th, two on 5th, one on 6th, and one on 12th, thirty-eight in all, of whom twenty-one died on board, and two on shore at Ascension. Though the crew of the *Bristol* included many young men fresh from England, and consequently quite unacclimatised, not a single case of the disease occurred in anyone who had not been exposed in the *Isis*. Here the *Bristol* was a healthy locality, for none of her crew had been attacked with yellow fever before this outbreak, nor were any subsequently.

But the same immunity is found, during the healthy season, in localities on shore where yellow fever springs up when the causes of that disease come into operation. Thus the Military Hospital at Barbadoes, which is close to the shore at the south-west part of the island, has some low ground just outside the enclosure, on the land side, where water collects after heavy rain, and the drainage thirty years ago being inadequate, a marshy spot of some extent was formed. As this was drying up, yellow fever appeared among the persons exposed to the emanations on several occasions. On 16th Nov. 1852, H.M.S. *Dauntless* arrived at Barbadoes from St. Thomas, with yellow fever on board, having lost several men during the passage; the sick were sent to the Military Hospital on 17th, part of the crew was landed, and part kept on board; the latter continued to supply fresh cases, which were sent to the hospital as they occurred, while the men on shore soon ceased to be attacked. Altogether, from Nov. 17th to Jan. 5th, one hundred and

* *Transactions* of Epidemiological Society, vol. iii, p. 481, and *Report on the Health of the Navy for* 1866, p. 226.

fifty-seven officers and men were treated in the Military Hospital, where sixty-five died. The fever cases among the men were mixed indiscriminately with the sick of the troops, yet none of these contracted yellow fever.*

The military cantonment at Newcastle, in Jamaica, is placed on a sharp ridge about 4,000 feet above the sea. The crest is so narrow in many places that there is room for a single hut only; at others it opens out sufficiently to receive two or more. The extreme length of the cantonment is only 800 yards, and the difference of level between the upper and lower extremities is about 650 feet. Yellow fever appeared among the troops here in the middle of September 1856, when it was prevalent on the low ground, and continued into December. The disease was confined to three localities, which alternated with four others in which there was much less fever, and of a much milder description, though there was uninterrupted communication from one end of the cantonment to the other daily. Including men, women, and children, there were 109 attacks of fever, and 45 deaths, of which 80 attacks and 41 deaths occurred among the people in the three unhealthy localities. The remarkable manner in which the unhealthy localities alternated with those where little fever occurred is very striking, notwithstanding the free communication that went on, and it showed the application of the cause of the disease must have been equally localised. During the epidemic 156 men were employed in the fever wards assisting the attendants, for periods of twenty-four hours, some of them twice, or oftener; altogether there were 210 such instances of exposure to the emanations from the sick in the fever wards; of these persons only eight were attacked with fever subsequently, of whom three died (two of these lived in the most unhealthy rooms at the station, and in the third, 32 days elapsed from his attendance to his attack); while of the remainder of the troops in the cantonment at the commencement of the outbreak, amounting to 523, there were 89 attacked with fever, of whom 38 died, giving a proportion of 5.1 per cent. of attacks, and 1.9 per cent. of deaths, for those exposed to the sick, against 17.0 per cent. and 7.3 per cent. respectively for those who did not come near them.† These instances illustrate the positions laid down above; there is a vast mass of similar facts in the literature of yellow fever, derived from nearly every country in which it

* Milroy, in *Lancet*, vol. i, 1861, p. 358.

† For full details see *British and Foreign Med.-Chir. Rev.*, vol. xxiv, p. 445.

has appeared, which can only be alluded to on this occasion, but which goes to prove that when the influence of locality is removed yellow fever ceases to appear, however numerous may be the sick, or however close may be the communication between them and those around them.

There is one case which requires notice here and has been held to establish the communication of yellow fever from man to man, in a position free from suspicion of the existence of local causes of the disease. I allude to one of the incidents connected with the *Anne Marie* at St. Nazaire in 1861. This vessel arrived there in July from Havanna with a cargo of sugar, having had nine cases of fever during the passage, two of them fatal. Some days after she commenced to discharge, yellow fever appeared among the people employed in this work, and among a number of others who belonged to other vessels lying in the vicinity. Some of the latter left St. Nazaire while the disease was in the incubation stage, and it became developed only after they reached their destination. A Dr. Chaillon who lived at Montori, 4½ miles (7 kilometres) from St. Nazaire, and had not been to the latter since the arrival of the *Anne Marie*, visited several of these men, and on the last occasion, on 11th August, finding the patient suffering from cramps, he remained with him and rubbed his limbs for nearly an hour. This gentleman, aged 42, though robust and active, was of a highly nervous temperament, very impressionable, and had a terrible dread of the risk he incurred by visiting these yellow fever cases. On 13th Aug., while 10 miles from Montori, he was attacked with intense headache and felt so unable to proceed, that he left his conveyance, and lay down by the wayside, until some of the people at work in the neighbourhood came to his assistance. He then made one or more visits, and returned home, the headache continuing very severe, with pain in loins, weariness in limbs, and he passed a very disturbed night, with delirium and occasional bilious vomiting. On the 14th he was somewhat better; he took a purgative, which operated very powerfully until the night of 15th, causing great exhaustion; he had himself bled on 14th to from 24 to 35 ounces, and took 1½ grammes of quinine. On 15th there was little fever, but in the night (the regular tertian period) there was much restlessness, and the eyes were slightly yellow. On 16th the yellowness became more distinct; during the night he had a little red wine and water, which was vomited soon after, and then had a blackish violet colour, and an execrable taste (*goût exécrable*). On 17th there was stupor, convulsive move-

ments of mouth and hands, more yellowness, petechiæ over forehead, arms, and legs. No urine was passed since previous night. Death took place at 11.30 a.m. Throughout the attack the overwhelming presentiment that his connection with the yellow fever cases would cause his death was ever present, and undoubtedly had the most injurious influence on its course.*

Pernicious intermittents are frequent in the country about St. Nazaire, and in August 1861, when the heat of the weather was almost tropical, intermittents were generally attended with bilious symptoms, and displayed a greater obstinacy than during the preceding years.† And before Dr. Chaillon's case can be received as one of yellow fever, the possibility of its having been merely a bilious intermittent must be excluded. The condition of the urine, which would have cleared up the difficulty, was not observed; the vomit, which has been taken as black vomit mixed with the urine, was described as having an execrable taste, which would lead to the inference that the dark colour it presented was due, not to black vomit, which, if not acid, has no prominent taste, but to bile, and the course of the disease otherwise was not incompatible with its having been a bilious intermittent, aggravated by the mental condition of the patient. It cannot be accepted, therefore, as a case of yellow fever, and does not establish the fact that yellow fever can be communicated from a person labouring under it to a healthy person, which experience elsewhere had already shown. M. Melier himself seems to have been doubtful of the nature of this case, for he says, "If it be not permissible to be altogether so explicit as to the propagation by sick, it must be admitted that the fact, it may almost be called the *experiment* of Chaillon, gives to this second proposition a high degree of probability, not to say of certainty."‡

Yellow fever, then, being clearly connected with locality, its exciting cause must be an emanation given off at such places, but whether this be gaseous, or consist of minute organisms, has not been ascertained. There are a good many facts, however, which throw some light on the condition under which this emanation arises, a right appreciation of which is of great importance as tending to place on a clearer basis several of the disputed points in the etiology of the disease. The first of these is whether the disease be really endemic in any country, and if not truly endemic,

* Melier, in *Mémoires de l'Académie de Médecine*, vol. xxvi. Letters of Madame Chaillon, Drs. Legoff, and Durand, p. 160 to 164.

† *Ibid.* Statements of De Guallouzo, pp. 129, 146. ‡ *Loc. cit.*, p. 96.

whether it may appear in a country from time to time without importation from some other in which it was already existing. The great frequency of yellow fever formerly in the West India Islands and around the shores of the Gulf of Mexico, led many authors to state that the disease was endemic in that region, though later experience has shown that it is occasionally absent for years from places where it had previously been nearly an annual visitant. The tropical part of the West Coast of Africa, also, has been commonly accepted as a perennial source of the disease, and Chisholm and Fegur attributed the epidemics they encountered in the West Indies to importation from that part of the world. The disease, however, was much less frequently met with in Africa than in the West Indies; thus there were epidemics at Sierra Leone in 1823, 1829, 1837, 1847, 1859, 1865, and 1872, and there was a single case of yellow fever there in 1827, another in 1835, and three in 1845, the year in which the *Delair* suffered from this disease. As the accounts of the commencement of the first four of these epidemics are pretty full, as well as of the cases in 1845, they afford some definite proof on the questions submitted above. Up to 1847, inclusive, the yellow fever in each of the epidemics appeared first at Sierra Leone, and in the three former was preceded by a considerable aggravation of the ordinary remittent fever among the crews of the various trading vessels which were distributed in the neighbouring rivers, collecting their cargoes of wood or other produce. Various vessels were supposed to have imported the disease, but, on examination, each of these was proved to have arrived in the colony in good health, and to have commenced to suffer some time after only, when the general sickness was beginning to show itself. In 1847 there was no suspicion of importation; the first case was that of the colonial surveyor, a gentleman who arrived in the colony from England on 3rd February, in the same vessel with myself. He was attacked on 29th June, and died on 3rd July, quite yellow, and having had black vomit. The second case, a person who had no communication with the first, was attacked about 11th or 12th July, and died on 16th. The third case occurred on 13th July, and died on 18th. There had been no communication between this and either of those that preceded it. The colony had been healthy until June. During that month, fever of the remittent form had shown itself in an aggravated form among some deserters from the shipping, and among a number of Spaniards and Brazilians who had been brought to Free-

town in captured slavers some time before; but in none of these cases had black vomit been met with, and, though some of them became yellow, these attacks had the usual duration and character of remittent fever.

The epidemic of 1823 extended to many men-of-war and other vessels, and embraced Ascension on the one hand, while one case at least occurred at Bathurst, on the Gambia, in an officer who had just arrived from Sierra Leone. That of 1829 commenced at the latter station in the early months; many ships were then affected, and it extended to Fernando Po, in the Bight of Biafra; in 1830, Goree and St. Louis, on the Senegal, were attacked. The epidemic of 1837 also commenced in the early months at Sierra Leone; the first case occurred at Bathurst on 24th June, and an epidemic followed, commencing about 25th July; and in August it broke out at Goree. At St. Louis there were a few suspicious cases, which were considered as marsh fever, influenced by the epidemic condition then existing. At the end of 1837, and in the early months of 1838, the yellow fever reappeared at Sierra Leone, and several ships were affected, and, after the arrival of some of them at Ascension, an epidemic appeared there. The epidemic of 1847 seems to have been confined to Sierra Leone and its immediate neighbourhood, no other place on the Coast of Africa, that I have heard of, having been affected; this year, however, as well as in 1837, there were epidemic outbreaks of yellow fever at several points in the West Indies, and on the neighbouring continents, so that the disposition to the disease at the time was really widely extended.

These facts show that from 1823 to 1847, yellow fever could not be considered as endemic on the tropical part of the West Coast of Africa, and yet that it appeared at Sierra Leone, and became epidemic four times at intervals of a few years, without importation from other places. It is obvious, therefore, that factors requisite for the production of the disease must have become active there, without introduction from elsewhere by shipping or sick; and this being established for Sierra Leone, when the conditions of the question had not been complicated by previous arrivals of ships from unhealthy localities, what is there to enable us to say that the same may not take place at the places on the same coast, where the same general climatic conditions exist? This has actually occurred: the British settlements on the Gambia were not affected in 1849 or 1830, but on the 13th June of the latter year yellow fever appeared suddenly in force, without having been preceded by the arrival of any vessel

for a considerable period, and spread over the neighbouring part of the mainland, reaching St. Louis on 4th August, where it caused great mortality.* The epidemic which commenced at Goree about August 1878, and which has extended very widely through the province of Senegal, seems to have arisen in the same manner; for there was no yellow fever at Bathurst in 1878, and apparently very little at Sierra Leone, the only notice of which I have seen being the occurrence of three cases in March, in black soldiers, all of whom recovered.

Again, when the sporadic cases are considered, they show that the factors may be in operation over a very extensive space about the same time, without any question of importation from one point to another, and yet, at most of them, not in sufficient form to generate an epidemic. Thus, in 1845, the *Eclair* had been at anchor off the Seabar, one of the mouths of the Sherboro river, south of Sierra Leone, from the beginning of March to nearly the end of June, with but little intermission. While here her boats were sent into the river on several occasions; cases of ordinary remittent fever appeared among the crews of these in April and May, but on 22nd May, a stoker, who had not been out of the ship, was attacked with fever, of which he died after five days' illness; the symptoms are not detailed sufficiently to enable the case to be recognised, with certainty, as one of yellow fever, but it was a very suspicious one. Others followed on 4th and 5th June, in one of which the patient became yellow, and in another incipient black vomit was found in the stomach after death, but these had been up the river from 22nd to 28th May.† The *Eclair* left at the end of June, arrived at Sierra Leone on 4th July, and finally left that place on 23rd July. While lying here her crew had leave. The first case of fever among them, since the ship's return to Freetown, occurred on 19th July, this terminated fatally on 27th, on board, and seems to have been the ordinary remittent. A second case occurred on 21st, which was sent to the Military hospital, it also was the ordinary remittent, and recovered. A third case, attacked on 19th, but reported on 22nd only, died on board on eighth day, having had dark grumous vomiting shortly before. A fourth case was placed on the sick list on 23rd, but had been unwell for some days previously; this appears to have been ordinary remittent. All these men seem to

* *De la Fievre au Senegal*, par L. J. B. Bérenger Féraud, pp. 116, 158.

† Bryson, *Account of the Origin, Spread, and Decline of the Epidemic Fever of Sierra Leone*, pp. 98 to 100.

have been on shore for some days. A fifth case occurred on 29th, and terminated fatally on 7th August, without yellowness or black vomit; and a sixth, put on the list the same day, died on 17th August, with a dusky colour of skin, and some frothy dark fluid was found in stomach.*

Up to the time of the *Eclair's* leaving Sierra Leone there had been no trace of yellow fever in the colony; but on 6th or 7th August, a clerk named Pringle, who had been in Freetown for some time, had ague; this passed into yellow fever, and he died on 16th, with black vomit. Mr. Babbage, master of the *Ceylon*, an American trader, which arrived on 28th July, from the Gambia and the coast to the northward, was attacked on 15th August, had black vomit, and died on 19th; and, lastly, a seamen of H.M.S. *Star*, named Elliot, who had arrived at Freetown on 2nd August, with six other white men, in an empty slaver from the eastward, was admitted into the Military hospital on 16th August with fever, of which he died on 20th, yellow, and having had black vomit. The officer of Elliot's party was attacked with ordinary remittent on 15th August, the other five white men embarked in H.M.S. *Actæon* on 17th August, where all had fever, and one of them died, but the form is not recorded.† It appears also that, in the last quarter of 1845, a corporal of the Infantry of the Marine was attacked with yellow fever at Goree, which proved fatal.‡

If, now, these facts be analysed, it is seen that up to and during the stay of the *Eclair*, no yellow fever occurred at Sierra Leone; the first case occurred in a resident, at least fourteen days after she left, and this was followed by two others on 15th and 16th August, in persons who arrived from sea, and from different points of the coast, after she left; the crew of the *Eclair*, therefore, could not have received the fever from exposure to *preceding cases* at Sierra Leone on the one hand; nor could the cases which appeared there in August be connected with her visit in July. Whether the case of the stoker in May, then, be considered as the first indication of the disease in her, or whether it be held that that only appeared after she left Sierra Leone finally, it is clear that it arose without previous communication with persons labouring under it, as it has been shown to do at Sierra Leone and Goree on previous occasions, and as actually occurred at both places in 1845. As bearing on

* Bryson, *Account of the Origin, Spread, and Decline of the Epidemic Fever of Sierra Leone*, p. 110.

† Bryson, *Climate and Principal Diseases of the African Station*, p. 170.

‡ Bérenger Féraud, *loc. cit.*, p. 120.

this question, too, it is worthy of remark here that the case at Goree appeared during the last quarter of the year, the time when the epidemic which broke out at Boavista after the *Eclair's* departure was prevailing in that island, which is the nearest of the Cape de Verd group to Goree, and only about 300 English miles from it.

The general outcome of all these facts is that from time to time there is an increased activity of those factors which lead to yellow fever, over a varying extent of the earth's surface, under the influence of which that disease becomes developed where local circumstances are suitable. The latter embrace the state of the weather, the condition of particular spots, often very limited in extent, and the emanation which engenders the disease in individuals exposed to it, while there are many points in their immediate vicinity where they may remain without risk, if only they avoid the emanation in question. The determining condition seems to be a certain amount of moisture of the soil at the point; whether supplied by rain, or from subsoil sources, is immaterial. Too much water obviates the result; whether by checking the formation of the emanation, or preventing its diffusion, has not been determined; while too little seems practically to stop its formation altogether. Thus, while it is found at Sierra Leone that yellow fever epidemics have fallen on years when the rainfall has been low for that place, and even then the disease has been met with before the heavy rains set in, or during a break in these which occurs pretty regularly in the end of July and early in August, or after the wet season has nearly terminated, in all the epidemics heavy and continued rain has put a stop to the yellow form of fever, and the ordinary remittent has taken its place. On the other hand, their vicinity to the desert ensures Goree and Senegal a much drier climate in ordinary years, and it has been remarked that their epidemics have all fallen in years more than usually wet.* At Boavista it was only after heavy rain, and when the pools resulting therefrom were being dried up under the action of the sun, that the fever became epidemic. The same combination is observed in the West Indies, a certain amount of moisture is always present when the disease springs up, and heavy rain for the time being seems to suspend the influence of the emanation.

What applies to localities on shore applies also to ships. Unless a vessel have her hold in a foul condition, she will not become the source of an epidemic; numbers of her crew

* Bérenger Féraud, *loc. cit.*, p. 298.

may be exposed to the cause of the disease on shore, and embark while it is in the incubative stage, but it is found to cease with them within the recognised period of incubation, and none become affected who had not been exposed in the sickly locality; but if, as in the *Eclair*, the hold be foul, it may supply the requisite conditions for the generation of the miasma under the influence of the pandemic factors, and a most serious outbreak may arise, while neighbouring ships, or even the shore itself, may remain quite free from any trace of the disease. A good illustration of this is afforded by the United States steamship *Susquehanna* in 1858. This ship, while at Spezzia in 1857, received orders to proceed to San Juan de Nicaragua, and left on 22nd October. After touching at Genoa, Madeira, and Key West (where there had been no yellow fever since 1854), she reached San Juan early in December, with her crew in perfect health. She seems to have anchored inside the harbour, where the shipping are within the range of the malaria from the swampy ground surrounding it, and in about ten days intermittent fever began to show itself, then remittent, and diarrhœa, cholera morbus, and scorbutic complaints were not infrequent. This state of things continued until 20th March, when the first fatal case of fever occurred. The ship was then taken outside, and cruized off and on for four days, then anchored sufficiently far off to be out of the reach of malaria; but the fever increased notwithstanding, and the sick list rose to thirty. It was now determined to proceed to the northward, and the ship left on 1st April, and reached Jamaica on 5th. On 6th and 7th, eighty-five cases of yellow fever were landed from her at the Naval Hospital at Port Royal, of whom twenty-three died. The *Susquehanna* left Jamaica for New York on 8th, and arrived there on 15th, fifty more cases of fever having been placed on the sick list during the passage. All the crew were ultimately removed from her, and she remained nearly three months with no one on board. At the end of this period people employed to remove her stores were again attacked with yellow fever. The surgeon of the *Susquehanna* had not heard of any yellow fever at San Juan while she was there, and though the other ships in port at the time had intermittent and remittent, none of them had any yellow fever.* There was evidently an active source of the disease in the *Susquehanna* herself; her hold contained the

* Report on fever in the United States steamship *Susquehanna*, by R. J. Macroom, M.D., in *American Journal of Medical Science*, vol. xxxvi, pp. 324-28.

soil suited for elaborating the emanation, and the other factor being supplied during her stay at St. Juan, it began to be evolved, with the result mentioned above; while, both on shore and in the rest of the shipping, for want of a suitable soil, the general factor proved inoperative. The sick from the *Susquehanna* were landed at Port Royal during the healthy season, and the disease died out with them.

A ship with a local cause for yellow fever in her may then, as in the case of the *Susquehanna*, retain the power of generating this for many months, and in that time may proceed from the place where she originally acquired it to a distant country, where those who come within the range of the emanations from her interior are liable to take the disease. Two questions, both important, but very different in their bearing, arise here in connection with the new locality, which require consideration, viz., First, to what distance from the vessel can the emanation prove effective in exciting the fever? Second, should the emanation meet with a suitable soil, can it fertilise it so as to make it give off a similar emanation capable of producing the disease?

The facts in my possession bearing on the first question are not very numerous. One instance occurred at the quarantine station at Pomegues, near Marseilles, in September 1821—a place perfectly healthy and isolated. There were forty vessels in it at the time; these were arranged round the port, their bows towards the centre and their sterns towards the shore, with a clear space between each two, and, being in quarantine, they had no communication with each other. The *Nicolina* arrived, having had some yellow fever on her passage, and was placed in quarantine. On 8th September her hatches were opened, and on the 11th the first attacks of yellow fever took place in the neighbouring vessels, which had previously been healthy. There were twenty-seven attacked, of whom seven died. According to a plan of the port, with the positions of the vessels upon it, the disease extended to the second vessel to the left of the *Nicolina*, a distance of 24 yards, and to the third on her right, a distance of 36 yards, the intermediate ships on both sides being all affected. And there was a single case in the seventh vessel to the right, at a distance of 70 yards, there being three vessels between this and the third which were unaffected. The wind was supposed to have carried the emanation from the *Nicolino* to the other vessels.* A plan of the dock at St. Nazaire, with the positions of the *Anne Marie* and the various vessels

* Melier, *loc. cit.*, p. 92, and plan in appendix.

that suffered, accompanies M. Melier's paper, the most distant of these, the *Cormoran*, had two cases, both fatal. These men were said never to have been on board the *Anne Marie*, though it is not mentioned whether they had been nearer her than their own ship, which lay at a distance of 187 yards. A mason who wrought at a point on the opposite side of the dock from the *Anne Marie*, and distant from her 284 yards, was attacked very early with yellow fever, of which he died. It was supposed this man had not been nearer that ship than the position where he was employed, but there seemed to be some uncertainty on this point.* In the outbreak at Swansea, in connection with the *Hecla* in 1865, described by Dr. Buchanan, two cases occurred on one side at 150 yards from the ship, and on the other side one case was 200 yards from her; but as a quantity of copper ore which she had brought from Cuba was there in a yard between her and the latter, it is possible it might have been the source from which the emanation was given off, which would reduce the distance to about 150 yards. A few days previous to the occurrence of the cases at these opposite points the wind had blown from the vessel towards them.† In 1861 three men of H.M.S. *Rinaldo* (last from England) contracted yellow fever at Halifax, Nova Scotia, which seems attributable to the same cause. H.M.S. *Spiteful* had arrived from the West Indies on 16th August with thirty-four cases of yellow fever on board. The healthy part of her crew were removed on shore to the dockyard, and the sick to a hulk in the harbour, while both parties and the vessel herself were placed in quarantine, as regarded the rest of the squadron. The *Rinaldo* was in the harbour at the time, and remained until 23rd, when she sailed for New York. On 27th August a boy was attacked with what proved to be yellow fever. On 28th a stoker was attacked, and on 29th another stoker. The first and last died, the second recovered. The boy was one of the dingey's crew, and was constantly employed going to and fro between the ship and the dockyard. The stoker attacked first was employed in the dockyard; of course returning to his ship every night. He denied having had any communication with the *Spiteful's* people. The other was a patient in the temporary hospital attached to the dockyard until the day previous to the ship's sailing.‡ Unfortunately there is no account of the relative positions of the *Spiteful* and the *Rinaldo*, or of

* Melier, *loc. cit.*, p. 125.
† Eighth Report of Medical Officer of Privy Council, pp. 449, 457, 468.
‡ *Report on the Health of the Navy for* 1861, p. 68.

the distance of the former from the temporary hospital. The *Spiteful* was admitted to pratique on 24th August, and on the 26th her holds were commenced to be cleared; that she continued to have an active cause of the disease in her soon became apparent, for on 31st one of her people, and next day two more, were attacked with it, all of whom died.* These facts show that the emanations from a vessel may be carried by the wind to considerable distances, and in sufficient quantity to excite the most pronounced forms of yellow fever.

Another mode of diffusion which has been much dwelt on requires notice here, viz., that by clothing, or other porous materials, which have been exposed in localities where the emanation was produced. Reference is frequently made to the behaviour of such articles when subjected to the action of odorous substances as illustrative of this point; but, like many other analogies, this is apt to lead to erroneous views, for the odoriferous element in all these being volatile, first passes into a state of vapour, and being subsequently condensed on the clothing, etc., a considerable quantity may accumulate, and these articles will then continue to give off the odour, so long as any of this matter remains for evaporation. As regards the emanation causing yellow fever, it must either be gaseous, or particulate; if the former, it may pass into the interstices of the dress or other porous material, but in no greater proportion than exists in the air in which it is diffused; if the latter, portions might be detained in the porous material, as a plug of cotton seems to detain fine particles, but the quantity so collected can never be considerable, unless the porous material has been exposed for some time to a current of air containing such particles, and in a thickness sufficiently reduced to permit of the bulk of the current passing through. It would appear, then, that such articles may receive and retain a certain portion of the emanation, and the question comes to be, is this sufficient to develope yellow fever in persons who have not been exposed to it at its original source. Several American authors adduce instances of boxes of clothing packed at New Orleans, causing the disease in those who opened them in New York; but they are not of frequent occurrence, and the every-day experience of men living among yellow fever cases, leads most of them to conclude that the amount of disease diffused in this way is infinitesimally small. It must be borne in mind that these remarks refer exclusively to the

* Slayter, *Transactions* of the Epidemiological Society, vol. i, p. 356.

emanation from the soil, or from similar sources of the disease in ships, and not to an infective material from the persons of the sick, supposed to be conveyed in their personal clothing and bedding, there being no small amount of evidence to show that, away from a locality where the terrestrial emanation is in operation, exposure to such articles fails to produce the disease.

Coming now to the second question submitted above, which is, in fact, whether the local conditions, giving rise to an epidemic, can be produced by importation, either by ships or fomites, there is, as most people are aware, a great division of opinion, due, in the main, to the parties concerned endeavouring to explain the extended operation of nature they deal with from too limited a view of the facts, and from insufficient analysis of the evidence at their disposal. If the facts connected with the appearance and progress of yellow fever for several years be collocated, it is found that, at its periods of epidemic activity, it has not been confined to one or two localities where it is commonly met with, but has extended far beyond these, sometimes in one direction, sometimes in another; the Atlantic Coast of the United States, and the Valley of the Mississippi, are two of these. In such extratropical extensions, the disease, as a rule, appears earlier in the season at the more southern points, and later at those more to the north; and persons from the former, proceeding north to avoid it, but who may have imbibed the poison before leaving, may arrive at their destination verging upon fever, or actually labouring under it, sometime before the conditions have been matured for the generation of the poison on the spot. When this takes place, and the disease manifests itself among the residents at the place subsequently, many think the evidence of importation complete. In the same way, should a ship, with the cause of this disease active in her, arrive at such a place, the subsequent appearance of the fever in the locality has been attributed to importation by her. Are these conclusions borne out by a full consideration of all the facts concerning the origin of yellow fever now available? I think not. The evidence concerning an outbreak at any given point is often so complicated that it is impossible to arrive at a trustworthy conclusion from that alone, and it becomes necessary to take other evidence, obtained at the same place at another time when the same complication may not have existed, or to have recourse to that from some other point, where the facts bearing on the question may have differed sufficiently to permit of those which

were not essential to the result being eliminated, and so permit of the proper inference being drawn from those that remain.

To give an illustration; in 1854 yellow fever was at Savanna, Charlestown, Portsmouth, and Norfolk, all on the Atlantic Coast of the United States; in 1855 it did not reappear at the two former, but at Portsmouth and Norfolk, where there had been a trace only the previous year, there was a severe epidemic; in 1856, Charlestown was the only one of the four places mentioned where the disease was active again, but this year there were upwards of 300 cases at various points around the harbour of New York; at the same time there was an epidemic in Bermuda, and great activity of the disease in the West Indies; and in Europe there were the first indications of the severe epidemic which occurred at Lisbon the following year. Dr. Vanderpool, health officer, stated there were a good many vessels from yellow fever localities in quarantine at New York when the outbreak commenced, several of which had had the disease on board. These were anchored near some of the districts on shore where the disease subsequently made its appearance among persons who had had no immediate connection with them, and he attributed its appearance to the emanation carried from those ships by the wind, and to portions of it adhering to various articles thrown overboard, which drifted to points on the shore near which the disease showed itself. Among other places where it sprang up was Governor's Island, the Military Station in the upper harbour, where it was confined to a fort at the south-east corner, which looks towards the quarantine station, upwards of five miles from it, but about a mile only from the nearest point on the neighbouring shore where the disease was met with.* Heavy rain had occurred some time before the yellow fever appeared on shore, and it was as this was drying up that it broke out. Now there is such a combination of circumstances here, that the influence of one cannot be separated from that of another; but when it is mentioned that a second outbreak occurred on Governor's Island in 1870 (in which there were 157 attacks, 49 of them fatal), when the disease was confined to another part of it, leaving the people in the fort it attacked in 1856, and many others untouched,† and where there seem to have been no vessels in quarantine with yellow fever on board, much of

* Abstract of Report in *American Journal of Medical Science*, xxxiv, p. 471, *et seq.*

† Sternberg, *Ibid.*, lx, 398, *et seq.*

the difficulty is removed, and factors thought to have proved so active in one instance, were found non-existent in the other. If, from New York we turn to Bermuda, it is found the epidemic there, in 1856, commenced at distinct points on the shore, well removed from shipping, and the first cases, in August, could not be connected with those in H.M.S. *Malacca*, which had been in quarantine with yellow fever on board, and had left the islands, with all her sick, on 28th June, or with another vessel, the *Margaret Messer*, from Demerara, which had had no sick on board at all;* nor could importation by ships be established in any of the epidemics which had appeared in Bermuda up to that date.† As factors similar to those which developed the yellow fever at New York in 1870 cannot be excluded in 1856, there is really no ground for assuming that the emanation from the vessels in quarantine, or from the refuse thrown overboard from them, had any influence in producing the local condition which gave rise to that which occurred in 1856; the experience of Bermuda, not differing much in latitude from New York, and, like it, subject to epidemics at intervals only, and which, in the majority of instances, occur at periods when yellow fever extends more or less along the Atlantic Coast of the United States, indicates the operation of some general factor unconnected with ships or traffic, as has already been shown to be the case on the West Coast of Africa; and it would appear, therefore, that beyond the tropics, as well as within them, yellow fever epidemics may, and do, arise without importation, either by ships or fomites. This conclusion, it may be said, is not incompatible with the active material of fomites fructifying in a suitable soil at some distant point, and so giving rise to an outbreak; but for the present there is no means of establishing this position, save by excluding the general factor already mentioned, and that our present knowledge indicates no means of effecting.

* Smart, *Transactions* of the Epidemiological Society, ii, p. 59.
† *Ib.*, pp. 30-68.

ON THE RELATION OF FILARIA SANGUINIS HOMINIS TO THE ENDEMIC DISEASES OF INDIA.

By SIR JOSEPH FAYRER, K.C.S.I., M.D., F.R.S.

(*Read: Feb. 5th,* 1879.)

In this paper I propose to draw attention to some aspects of an important helminthological discovery, and also to certain points on which, in its relation to endemic disease in India and other tropical and sub-tropical regions, further information may be anticipated.

I must say at once that I have nothing original of my own to communicate, but deal entirely with the work of others. I am glad to have this opportunity of testifying how deeply medical science is indebted to our brethren at home and abroad for their researches. I know of nothing that more practically illustrates the scientific zeal of our profession than the subject I have to bring before you.

In March 1870 Dr. T. R. Lewis, of Her Majesty's British Medical Service, made known his discovery of certain microscopic nematodes in chylous urine, and suggested their probable causal relation to that disordered state of the lymphatic system. In the appendix to the Sixth Annual Report of the Sanitary Commissioner with the Government of India, 1869, published at Calcutta in 1870, he states that when examining the urine of a patient in Calcutta suffering from chyluria, he discovered amidst the fibro-albuminous matter in that fluid the embryos of a new round worm each time he repeated the examination, and he noted appearances that indicated the existence of a sheath in which it was enclosed. He at first thought that what he saw were detached filaments of a fungus, but movement revealed their real nature. As to their size, considering "that two hundred could pass abreast through a pin-hole, an orifice not exceeding one-fiftieth of an inch in diameter, it is not surprising that their existence had not been suspected". Lewis, I suppose, was not then aware that they had been

seen, under similar circumstances, by Wucherer, in Bahia, in 1866. This appears to be the earliest notice of their existence in India, and certainly it was an original discovery of the greatest importance in its bearing on hitherto unsuspected causes of certain diseases. In July 1872, when examining the blood of a native in the Calcutta Medical College Hospital, who was suffering from diarrhœa, Lewis detected nine nematoid worms, in a state of great activity, on a single slide. They were similar to those observed in chylous urine in March 1870. Specimens were sent to Dr. Parkes, who, with Mr. Busk, expressed an opinion that they belonged to filariæ: it had not hitherto been known that they were hæmatozoa. The name "Filaria sanguinis hominis" was given to them. Further examination of cases of chyluria proved that both in the blood and in the urine the filariæ were to be found, and also in morbid secretions. For example, in a case where chyluria was associated with granular eyelids and inflammation of the eyes, giving rise to a milky secretion from the corners of the eyelids, filariæ were found in the exudation.

Lewis gives a rough estimate of the number of filariæ at 140,000, being an allowance of two individuals in each drop of the ten pounds of blood possessed by a person of one hundred pounds weight. This, if the parasites pervade the whole blood, as they probably do, is, I should think, considerably below the mark.

The creature is very minute, and apt to be overlooked without long, careful, and patient examination. The power employed should be sufficiently high to define a red blood-globule. The diameter of filaria sanguinis hominis is about $\frac{1}{3500}$ in. Some are larger, some smaller: the largest is $\frac{1}{3000}$ by $\frac{1}{68}$ in.; the smallest $\frac{1}{7000}$ by $\frac{1}{125}$ in.

Filaria sanguinis hominis belongs to a family that includes many other entozoa prejudicial to man and animals—ascaris, oxyuris, dochmius, speiroptera, anguillula (the diarrhœa worm of Cochin China), dracunculus, trichina. It is allied to the two last mentioned, and their larval forms are somewhat alike, though perfectly distinguishable. During the first few hours "after removal from the body, the filaria is in constant motion, coiling and uncoiling itself, and lashing the blood-corpuscles about in all directions". It has the power of retracting the head and tail. When recent it is smooth and transparent; some have a granular appearance; a spot, like an oral aperture, sometimes appears. As they become inactive they lose their transparency; if a little spirit be added they preserve their outline, otherwise they

quickly disappear. They are capable of retreating within the delicate sheath by which they are surrounded. Under careful examination, an oral and an anal aperture, and an intestinal tube, can be made out.

The embryo being enclosed in a sheath, and having no visible boring apparatus, we may assume that its home is in the blood (or lymph), and that its movements are those of the circulation. With regard to the part taken by it in the production of disease, *post-mortem* evidence is much needed, not only with the view of determining what pathological changes take place, but of ascertaining the seat of the parent worm, and especially should search be made in and near the blood and lymph channels. Lewis describes the *post-mortem* appearances in the kidney thus: "It was more than usually lobulated, and on section several of the pyramids, especially near their apices, presented a smooth tallowy appearance suggestive of amyloid disease; no approach to the characteristic iodine reaction was obtained; translucent oil-like tubules, of a varicose appearance, were observed alongside of the uriniferous tubes, as if the vessels had been plugged." Boiling ether had no effect on them. No other morbid changes were found, but in every part of the kidney examined filariæ were found, as also on the branches of the renal artery, which were not, however, enlarged. The capillary vessels, from absence of good preparation, were not examined. Such is the course of the hæmatozoon into the urine. Worms of various sizes were found in the chylous urine, but no casts.

Chyluria is frequently associated with symptoms of other obscure diseases: diarrhœa, often of a persistent character, deafness, chronic ophthalmia. And here I may notice that in horses, and men too, in Africa and India, a filaria is occasionally found in the eyeball. I have several times seen horses blind from the presence of a living and moving nematode in the anterior chamber, which quickly induces corneal opacity. There are several other morbid conditions that I shall presently refer to.

The chyluria, due probably to the stoppage and rupture of lymphatic vessels, is doubtless very frequently associated with filaria; but we may not, I think, affirm that it is always so caused; other conditions may give rise to it. But in warm countries, or in cases of persons who have long resided in them, it is important to consider this probable origin, not only in chyluria, but in chronic diarrhœa, especially if associated with intermittent chyluria. It has been shown that the parasite may live for years without

causing any very palpable derangement of health, and that it does not undergo any development as long as it remains in the circulation. It may continue an abiding source of disease, which under certain conditions will develope itself, and what these conditions are we now seek to learn. This is an important subject on which we seem likely to add to our knowledge.

There are others who share the honour of the discovery with Lewis. The literature of the subject is already extensive. I can only refer to some of those who have contributed to it. Dr. Cobbold, who has himself added so largely to our knowledge, not only on this but all else connected with helminthology, gives a list of authors.

In 1843 Klenke found worms in the blood of a person suffering from vertigo, but as he did not describe them exactly, we cannot say if they were nematodes.

In 1868 (or 1866) the late Dr. Wucherer, of Bahia, published an account of a worm that he had detected in the urine of a person suffering from hæmato-chyluria. He found it when searching for bilharzia; and describes the minute nematodes as narrow at one and obtuse at the other extremity. He found them on subsequent examinations of chyluria, and they were always alive and active.

In 1868, Dr. Salisbury, in the United States, discovered a small entozoon in the bladder of a chylurious person. He described it as a new form, and called it Trichina cystica.

In July 1870, Dr. Cobbold, F.R.S., found a minute nematode and eggs in the urine of a little girl from Natal, suffering from bilharzia. He says that though numbers of fluke's eggs were passed daily with much blood, it never occurred to him that the nematode was hæmatozoal. The child's parents had told him that she had passed three small worms by the urethra long before, and he assumed that they were of urinary origin. Had he, as he remarks, examined the blood from a finger, Lewis's important discovery of microscopic hæmatozoa might have been anticipated.

In or about 1872, several "finds" were made in foreign countries. I cannot detail them, but they will be found in Dr. Cobbold's interesting paper in the *Proceedings* of the Linnean Society, October 1879, vol. xiv, No. 76, and in a paper in the *Veterinarian*, a translation of a memoir by Da Silva Lima, with appendix by Le Roy de Méricour; also in the August and September numbers of *Archives de Médecine Navale*, by Bourel Ronciere.

Up to 1872 the discoveries had reference only to the urine. In July 1872, however, Lewis discovered the nematode in

the blood under circumstances already described in a case of diarrhœa, and in the following October in that of a former chylurious person. The worms were identical. This very important discovery of the blood stage is, therefore, Lewis's, although the embryo had been discovered some time previously in the urine by Wucherer.

The parent worm was discovered still later by Dr. Bancroft in Australia. Thus we have separate discoveries of the different phases of filaria from widely distant quarters of the globe.

Dr. Manson, in China, ascertained that the mosquito is its intermediary host. To all much credit is due, and whether we call the parasite F. Wucherii, F. sanguinis hominis (Lewis), or F. Bancroftii, is unimportant. If the blood stage (which is by no means certain) be the most important phase of its genetic cycle, we may perhaps with advantage retain the Indian name; if the parent be the cause of most mischief, which may prove to be the case, then, perhaps, Bancroftii will be the most appropriate specific distinction. In any case, to each of these gentlemen is due the honour of a most important discovery.

To revert to the embryo. In 1874 Dr. Sonsino, in Egypt, when examining the blood of a young Egyptian Jew suffering from bilharzia hæmaturia, detected a minute filarial embryo, which he was good enough to demonstrate to me in Cairo when I was there in 1876. I wrote a notice of it in the *Lancet* as filaria sanguinis hominis Egyptiaca. It seemed probable that it was a variety, but there is, I believe, no doubt that it is identical with Lewis's species. Sonsino has also discovered a filaria embryo in the blood of the horse and of the crow. He is adding and will add to our knowledge of the entozoa of that part of the world.

In 1875, Dr. O'Neill, on the South Coast of Africa, found embryo nematodes in the exudation from the skin in a disease named by the negroes "craw-craw"; whilst Dr. Araujo detected it at Bahia in a negro suffering from the same disease; he called it "filaria dermithemica". F. Dos Santos found it, once only, in the blood of a person suffering from elephantiasis, in Bahia. Dr. Crevaux detected it in chyluria in Guadaloupe.

Certain free microscopic nematodes, very closely resembling filaria sanguinis hominis, if not identical, have been found in the potable waters of Rio (Agua di Carioca), by Dr. Magalhaes. Their genetic relation with filaria sanguinis hominis is not, I believe, quite established; but the indication is important,

as it points to the channel through which the parasite may find entrance to the human body.

Dr. Manson, of Amoy, has discovered, what had been already suggested by Dr. Bancroft in Australia, that the hæmatozoa are passively transferred to the stomach of the mosquito, and he has described the transformation they there undergo. A new and important *rôle* for this already obnoxious insect has thus been revealed, the full import of which remains still to be ascertained. It appears that the mosquito has a peculiar facility for ingesting filariæ, as the blood taken from their stomachs contains more parasites than that in the person they have fed on. Manson says that the embryo, after entering the mosquito's stomach, retains for a short time the appearances which characterise it in the human blood. But after a few hours changes begin, a double outline of body and marked striation appear, and again disappear. This is the first stage in metamorphosis, and occupies about thirty-six hours. It now enters on a sort of chrysalis condition, in which, though quiescent, it changes in form. A four-lipped mouth and an intestinal tract become apparent about the fourth or fifth day. The subsequent details are difficult to trace, but it is now one-fortieth to one-thirtieth of an inch in length. It now completes the mosquito phase, and is very active, "moving about with great facility and rapidity in the water into which it has emerged, equipped for independent life".

Lewis also found embryos in mosquitos in Calcutta, and has described them in the Bengal Asiatic Society's *Journal* of March 1878. He says that most of them came from pariah dogs, and they very closely resembled the human parasite. But he says that the mosquitos captured in a particular servant's house did contain human filariæ, and that he had the opportunity of observing all the stages of their growth; that whilst the stomach digests some, others perforate its walls, pass out, and undergo developmental changes in the thoracic and abdominal tissues of the insect. He thinks that it will be found that Manson's observations apply to India, but he does not, I think, feel convinced that the mosquito is the only intermediary host. It may turn out to be so, but it is to be noted that even the most advanced stage hitherto seen is still a very immature one, no trace of reproductive organs being distinguishable.

Dr. Cobbold, I believe, thinks we have sufficient information to establish the genetic relationship between filaria sanguinis hominis—the stomachal filaria of the mosquito—and the sexually mature filaria Bancroftii. This name is given

by Dr. Cobbold to the mature worm, in honour of its discoverer, who first found it in Australia in 1876. He sent specimens home, and they were verified by Drs. Cobbold and Roberts. Dr. Cobbold having detected, among specimens of microscopic hæmatozoa sent home by Bancroft, a single empty egg-covering, which corresponded with some he had observed in a Natal patient, suggested search for the parent, and Bancroft found it in a person suffering from lymphatic abscess of the arm in December 1876. Lewis made further search, and on August 7th, 1877, found two specimens in the blood-clot from a young Bengalee lad who had been operated on for nævoid elephantiasis. Carter had also found it in India in 1876 in a lymphatic abscess of the arm and in hydrocele of the spermatic cord. Drs. Araujo and F. Dos Santos and others also verified the discovery. Bancroft sent a specimen to Dr. Cobbold, who gave an account of its anatomy in the *Lancet* of October 1877, with figures. He only found the female, and gives the following dimensions. Mature: length, $3\frac{1}{2}$ in. Embryo: length, $\frac{1}{200}$ to $\frac{1}{125}$ in.; breadth, $\frac{1}{2000}$ to $\frac{1}{2500}$ in. Body capillary, smooth, uniform in thickness; head with a simple circular mouth, destitute of papillæ; neck narrow, about one-third of the width of the body; tail of female simple, bluntly pointed; reproductive outlet close to the head; anus immediately above the tip of the tail. A number of ova and embryos escaped from a mature worm during examination. Lewis also gave a description (and figures) in the *Lancet* of September 19, 1878, which very closely corresponds to that by Dr. Cobbold, and says: "It is possible that when the parasite discovered by Dr. Bancroft has been more definitely described, and its anatomy investigated, it may become evident that it is identical with the filaria sanguinis hominis." Of this I imagine there is now little if any doubt. I should add that Lewis noticed that the embryos that escaped from the mature worm detected in the blood-clot of nævoid elephantiasis were identical with embryos found in the blood of other parts of the body.

As regards the mode of entry into the human body, Manson says, speaking of the embryo *after* it has left the mosquito and escaped into water, "It is through this medium brought into contact with the tissues of man, and thus, either by piercing the integument, or, what is more probable, being swallowed, it works its way through the alimentary canal to its final resting place; arrived there, its development is perfected, fecundation is effected, and finally the embryo filariæ we meet with in the blood are

discharged in successive swarms and in countless numbers. In this way the genetic cycle is completed." With regard to a boring apparatus, Manson says, "I cannot say if the three or four papillæ round one extremity of the developed embryo constitute the perfected boring apparatus of the worm, or if it is a boring apparatus at all; but comparing this with what is found in other species, I think it very probable that it either is or will become the piercing apparatus."

I may not dwell longer on these details. They will be found in full in Dr. Manson's excellent paper in the Linnean Society's *Journal*, No. 75, vol. xiv.

I would just remark that whilst this is probably the right interpretation of what takes place, it cannot be regarded as perfectly established until further observation shall have confirmed the facts. This much, however, may be regarded as certain. The embryo filariæ may swarm in the blood, and be associated with or give rise to certain morbid conditions. They pass into the mosquito, or it may be into other intermediary hosts, where they undergo developmental changes in the tissues of the insect, and are at length set free in the water along with the larvæ. We cannot actually trace them further as they pass into man, but we have good grounds for believing that they do so through the medium of water, nor can we follow them through the further developmental changes that result in sexual maturity, though we know that the fully developed worm finds its home in the tissues of man, and then sheds its embryos into the circulating blood. How much and in what proportion the diseased conditions that result from their presence are due to the embryo, how much to the parent worm, we are still unable to say; but we have good ground for believing that *post mortem* examination will soon throw light on the subject, and on the precise localisation of the parent worm.

It is not within the scope of this paper to describe the diseases due to nematodes generally. I must restrict my remarks to those peculiar to filaria sanguinis hominis (Lewis).

Great advances have been made in our knowledge of this subject during the last four or five years in reference to the relation of this parasite to certain forms of tropical disease. It has been shown that disorders of the lymphatic system are most frequently associated with, if not caused by, the filaria; also nævoid and ordinary elephantiasis Arabum; perhaps also elephantiasis Græcorum,* chyluria, hæmaturia, hydrocele,

* I find in the *Lancet* of February 1st, 1879, that Dr. Bancroft has

and affections of the cord and testis, lymph varix and abscess, diarrhœa, fever, cachexia, deterioration of general health, certain skin diseases (craw-craw), deafness, eye disease. All these have been ascribed to it, though it is not asserted that they are *always* so caused. Being symptoms of impaired or impeded function they may be produced otherwise; but it is tolerably certain that we have now got one explanation of their origin in a hitherto unsuspected agency. The mechanical action, were there no other effect, might explain much that occurs by the plugging and obstruction of the nutritive channels. To say nothing of the direct effects on the blood itself, one can hardly conceive that these organisms should exist in the blood in any numbers without affecting it prejudicially; and yet we know that they have been found in the blood of persons who were apparently healthy. Of course it is difficult to define any absolute standard of perfect health, and there may be a considerable amount of cachexia, even in persons generally regarded as healthy, though perhaps, could we know their real condition, we might think otherwise. What the peculiar change in locality or action on the part of the hæmatozoon may be that determines the diseased condition we are still ignorant, and this is just one of the points that need elucidation.

We naturally ask if there be any analogy in the conduct of other entozoa in this respect, and in comparative helminthology also we look for guidance. Dracunculus causes little or no annoyance in its earlier stages, but in developing, changing place, and discharging its embryos the gravest results accrue. So trichina, which in its earlier stages is comparatively unfelt, may become the cause of fatal disease.

When the blood swarms with minute organisms, one might expect to find some evidence of their presence, either from mechanical interference with the blood or lymph circulation, or from *direct action on the blood itself*, or on the nerve-centres. Of the first we have an example in lymph-scrotum, elephantiasis, and chyluria; to the second I believe it is probable that several morbid states hitherto ascribed to other causes are due, and, judging from analogy, we may believe that this is so. We know how severely animals suffer from the presence in the blood of other minute organisms. Anthrax, for example, is produced by bacillus;

recently written to Dr. Cobbold that he has found filariæ in the blood taken from anæsthetic patches in the thigh of a European, aged forty-nine, in Brisbane, suffering from leprosy. Dr. Bancroft also says that he has examined the blood of two patients who formerly had filariæ associated with abscess of the arm; their blood is now free from hæmatozoa.

a cholera-like disease in pigs is produced by the migration of stephanurus, as pointed out by Dr. Cobbold; phthisis in sheep by strongylus filaria; and in man, as shown by Griesinger, the so-called and widely-spread Egyptian chlorosis owes its origin to Dochmius duodenalis (Leukhart), and that not only anæmia, but liver disease and a form of dysentery, are produced by the same parasite, which is not very unlike filaria sanguinis hominis. "Similar facts", says De Chaumont, "have been observed in Brazil, Arabia, Madagascar." It seems impossible but that in India the formidable affections caused by the dochmius should not be common. Manson and Lewis have shown that certain filariæ (F. sanguinolenta and F. immitis) infest dogs in China and India; they very closely resemble the human parasite, and give rise to certain diseases. Sonsino has demonstrated filariæ in the horse and crow in Egypt. In July 1872, Lewis found in the exudation pressed out from the mesenteric glands of a pariah dog, in Bengal, a small embryo nematode, that very closely resembled the filaria sanguinis hominis, and corresponded to filaria sanguinolenta of Rudolphi. They were found also in connexion with the walls of the stomach and œsophagus, as had been stated by Czernay and others; in the walls of the blood-vessels (especially of the thoracic aorta); in the interior of fibrous tumours of from the size of a pea to that of a filbert, or even walnut, on the walls and in the substance of the coats of the vessel, there might be seen a pitted, or sacculated, or atheromatous condition of the inner coat. There was also an enlargement and softening of the glanular structures near the base of the heart.

The tumours of the aorta afforded opportunity of studying the growth of the parasite from an early period up to maturity, as specimens of almost every stage of development may be found lodged in the walls of a single aorta. The parasite is found coiled up in the tumour. They are, immature, $\frac{1}{11}$ in. in length, $\frac{1}{180}$ in. in breadth; some smaller. When at this age they moult. Further changes take place, and, by the time the worm has become three-quarters of an inch long, reproductive organs can be made out. It now gradually acquires a pinkish colour, and, instead of occupying a small tumour alone, it joins other worms in a larger tumour, each occupying a different tunnel. Sometimes they make their way outside. When mature the worm is from three-quarters of an inch to one inch long, and one-fortieth of an inch broad. The female is two to three inches in length. They were also found in the thoracic glands and stomach.

The ova are oval, $\frac{1}{700}$ in. in size. When crushed an inactive embryo escaped. Lewis has a doubt as to the habitat of these embryos—whether they are in the moist earth, the water, or the intestinal tube of some other creature. We have thus the opportunity of watching the development of the sexually mature worm. Manson made similar investigations on Chinese dogs, and found F. sanguinolenta associated with F. immitis. The free embryos in the blood of such dogs are probably the progeny of either or both F. immitis and F. sanguinolenta. It is said that F. sanguinolenta is oviparous, whilst F. immitis is oviviparous. F. immitis is larger than F. sanguinolenta. It is found in the mature form in the heart of the dog, and its broods of embryos swarm in the blood. Time does not permit of my dwelling on these canine filariæ. I have only done so, so far, in regard to their analogy with the human parasite, which probably, in its mature form, leads a similar existence, shedding its broods of embryos into the blood-vessels or lymphatics.

Dr. Manson describes the following diseased conditions in dogs caused by filariæ:—F. sanguinolenta gives rise to three serious morbid states—(1) stricture of the œsophagus; (2) pleurisy; (3) paralysis of the hind legs, caused by the presence of the worm in the tissues about the œsophagus, causing tumours — tumours bursting into the pleura — plugging of the capillaries of spinal cord by ova, and other affections, that may be produced by them in the intestines, kidneys, or other viscera. The F. immitis, on the other hand, affects the heart and lungs. By arrest in the lungs they give rise to tuberculoid disease—at all events, an infiltration resembling miliary tuberculosis; but more frequently they manifest themselves in the heart's action by interfering with the integrity of the valves or the capacity of the pulmonary artery or its branches. Death not unfrequently results suddenly, or apnœa, failing circulation and respiration, ending in death. Such animals are short-winded, and liable to attacks of syncope; and one can readily imagine many other complications arising in animals so affected, and it is said that two-thirds of them are so in China, where they frequently are the subject of both filariæ. Their mere presence, even in great numbers, does not necessarily produce symptoms of disease, and this is very remarkable when we consider the demand these creatures must make on, and the change they may effect in, the nutritive fluids. It is interesting to notice the action of other allied forms in man: *e.g.*, trichina and dracunculus behave nearly

in the fashion of F. sanguinolenta in the dog; they migrate, pierce the tissues, develop, burst, and give exit to embryos which set up irritation, perhaps induce fever and other secondary affections, to say nothing of those caused by their immediate presence; and yet there is a period in their existence, too, during which there is little, if anything, to indicate their presence. Recently we have learned from the French physicians, Drs. Normand and Bavay, of new minute nematodes from Cochin China—the Anguillula stercoralis and Anguillula intestinalis; the former found in the stomach, the whole length of the intestine, pancreatic and gall ducts, 1 millimètre in length, 0.04 mm. broad; the latter in the hepatic ducts, the walls of the gall-bladder, less frequently than the former, but three times its length and sixty-five times its breadth. These are described as giving rise to dysenteric diarrhœa, which is very intractable, and often ends in marasmus. You will find a description of this parasite in the *Archives de Méd. Navale* of September 1878.

We have still much to learn about these human nematodes, especially in regard to their mode of entering into the body, their locality, habitat, and the way in which they become sexually mature, where and when fecundated, and where precisely the embryos are let loose among the tissues, and how they find their way into the blood and lymph-vessels, and the precise relation that these vessels bear to each other as carriers of the ova or the embryos. The subject is in the hands of competent observers, and we may look with confidence for a solution of much of what now seems obscure, especially from India, where *post-mortem* examinations apparently are more easily obtained than in China.

I have given a brief outline of the principal facts relating to the human blood filaria, and have alluded to some connected with its congeners that affect the lower animals, so far, at least, as they throw light on what is obscure in the human parasite. It remains to be shown what we already know and in what direction we are to look for further information.

I have said that filaria is frequently associated with, if not the actual cause of, chyluria and other disturbances of the lymphatic system. It is on this part of the economy, indeed, that its influence is specially exerted. Some years ago, in writing about nævoid elephantiasis, I said that I thought it probable that other forms of disease would prove to be due to filaria; and recent observations confirm the

prediction. I repeated the observation in 1876 when writing on Sonsino's discovery of the filaria in Egypt, and almost daily proof is forthcoming that in it is to be found the explanation of much that has hitherto been etiologically uncertain. I believe that we are still only on the threshold of our discoveries in regard to this form of helminthiasis. When we consider how much has already been traced to this parasite, it is impossible not to infer that more will be revealed.

Dr. Bancroft suggests that the following conditions are probably associated with filaria: chyluria, hæmaturia, anæmia, tuberculosis, hydrocele with milky fluid, varicocele, elastic tumours in the axilla and groin (helminthiasis elastica), lymph-vesicles bursting in the scrotum and abdomen, skin disease (craw-craw), acute orchitis, lymphangitis with fever, erysipelatous lymphangitis leading to hypertrophy of the skin (elephantoid fever in short), elephantiasis of the scrotum and leg, contracture of the lymphatic trunks with neuralgic pains, abscess of the scrotum, of the glands of the neck (like struma), of the lymphatics of the arm and thigh, intra-pelvic abscess, peculiar steatoma of the face, venous varix, cerebral abscess and other lesions of brain—a long list suggestive of mechanical interference with the free passage of the nutritive fluids.

Manson's experience in China, and that of Lewis, McLeod, McConnell, Ewart, Carter, Palmer, and others in India, show that elephantiasis, both nævoid and simple, is associated with filaria, and probably to these may be added other affections of the lymphatic system. My impression is that other morbid conditions, tropical cachexia, nervous disorders (paraplegia and others), inflammation of serous and gastro-intestinal mucous membranes, diarrhœa, and certain forms of dysentery may be due to it, directly or indirectly. Beriberi, anasarca, dropsy, albuminuria, hæmaturia, may, like chyluria, receive a new explanation. The vague chronic ailments that sometimes adhere to old residents in tropical or sub-tropical climates may in some instances depend on the presence of these hæmatozoa. There are certain forms of hydrocele, epididymitis, and inflammatory conditions of the spermatic cord and its appendages, peculiar to tropical climates, that long puzzled me as to their etiology, that now begin to appear more easily accounted for. The tendency of the blood to form fibrinous coagula while within the walls of the living vessels and heart; the embolisms, thromboses, cardiac and pulmonary arterial pluggings; the arterial, venous, and capillary obstructions resulting in death;

apnœa, gangrene, softenings, or ulcerations; that peculiar tendency in the blood of persons in malarial climates, notably Bengal, to form these fibrinous clots in the heart or pulmonary artery, so often fatal, may, too, in some measure, be referred to the same cause; not always surely, but possibly in some cases. I merely suggest it as worthy of consideration. The dogs in China die suddenly from the occurrence of interference with the heart's action due to filaria; whether to direct mechanical interference or to altered conditions of the blood I cannot say, but what occurs in the dog may, from a similar cause, occur in man; at all events, I know from experience that men do so die and suffer in Bengal—whether they are filarious or not remains to be shown. Had I known of the existence of filaria when I had such cases, I should certainly have looked for it; and may it not be possible that such conditions as result from obstructed capillaries, boils, carbuncles, local deaths of tissue, abscess, ulcers, whether of skin or other tissue, or of the viscera themselves, are so caused? The blood-vessels, too, may suffer from aneurism, varix, or some other condition of disease. In the dog they are so affected. So with stricture of the œsophagus, may we not, I repeat, in the present imperfect state of our knowledge, look for similar diseased conditions in man? Analogy warrants it, I think. Manson says that in the blood of 670 persons examined, of whom 195 were in apparently good health, he discovered filariæ 43 times associated with some morbid state, and 9 times coexistent with the appearance of perfect health. Here is the list of what he found:—

Elephantiasis of scrotum	3
„ of leg	1
Lymph-scrotum and chyluria	2
„ „ and elephantiasis	1
„ „ and inguinal varix	1
Lymphatic inguinal varix	7
Ditto, coexistent with ulcer of cornea	2
Hæmatemesis	1
Cataract	1
Ulcers of leg	4
Hydrocele	3
Hydrocele and rheumatism	1
„ and cataract	1
Rheumatism	1
Tumefaction of spleen	1
Fever and anasarca	1
Leprosy	2
Debility	1
Intermittent fever	2
Intermittent fever or spleen	1
Carried forward	37

Brought forward - - - - -	37
Intermittent fever with inflamed scrotum - -	1
Hydrocele and fever - - - - -	1
Chronic eczema - - - - - -	1
Internal hæmorrhoids - - - - -	1
Stricture of œsophagus - - - - -	1
Heart disease and ulcers - - - - -	1
Total - - -	43
Free from apparent disease - - - -	9
	52

It would seem that many forms of disease may be associated with, if not caused by, this parasite, in one or other of its genetic phases. It is necessary to be careful, lest, in the enthusiasm of a new discovery, too universal application be made of, and more ascribed to, it than is its due.

Important questions arise out of the preceding considerations :—

1. Whence does filaria come?
2. What can we do to prevent or remove it?

The second question will be best answered when we are able to reply satisfactorily to the first; when we know the origin, and are assured of all the genetic phases and the whole life-history, we shall be in a better position to deal with it. Prevention is the first desideratum, but when infection has occurred we need some effective parasiticide, and it is not improbable that such exists. Perhaps carbolic or sulphurous acid, arsenic, or, it may be, some of the remedies that have proved useful as antiperiodics, febrifuges, may have owed some of their virtues to their antihæmatozoal properties. Secondary consequences, such as are described in Manson's list, must be treated on ordinary medical and surgical principles, which at present I cannot stop to describe.

The area of distribution of these diseases is said to correspond with the geographical distribution of culex. Where the mosquito is rare the diseases of filaria are infrequent, and the reverse. Manson advocates this view, and is impressed with the importance of the part played by the mosquito as intermediary host and propagator of the parasite. He certainly has grounds both in fact and analogy for his belief. Dracunculus employs the microscopic crustacean cyclops as an intermediary host. A second stage is thus, like that of filaria, passed in another creature; they both find their way, through the medium of water, back to man. He adduces an authenticated instance of importation of the disease of elephantiasis by a single individual into Barbadoes. The mosquitos sucked his blood, and with it em-

bryo filariæ. They were thus disseminated, and the disease has been ever since endemic in the island where it was before unknown. I cannot affirm that the dissemination actually took place through the mosquito, but the rest of the story is authentic—the appearance in the island of elephantiasis on the advent of a particular individual. I believe Dr. Manson thinks the mosquito did it; if that be not the real explanation, I know no other. In any case, whatever part the mosquito may play, the water must be regarded with grave suspicion, and the greatest care observed in securing its purity; whilst in view of what we know of the mosquito, it is desirable that he should be kept from access to all water-supplies for either drinking or bathing, and that the water should be boiled before using. Sanitary science has already reduced the death-rate of our soldiers in India from 60 to 16 per 1,000. May it here achieve further triumphs. It will be well, at all events, not only to insist on pure water, as it always does, but to keep its eye on mosquitos and other possible intermediary hosts. The best way of getting rid of an entozoal pest is to interfere with its genetic cycle.

In concluding my remarks, I would say that I feel it is presumptuous to suggest any line of investigation. The discovery of the filaria and its relation to disease is so complete as far as it has gone that it seems hardly right to trespass on ground so originally and advantageously occupied. I know that I have most imperfectly represented the work already done, and the only justification I have for dealing with the subject at all is obedience to your behests, and that, having had considerable experience of the diseases ascribed to filaria, I am so far qualified to express my sense of the value of the light that has been thrown on a very obscure subject. I therefore esteem it no less a privilege than a duty to declare how much pathology is indebted for the knowledge gained. I am aware that the subject is still in its infancy, and that further research may modify much we now seem to know; but we are on a new and original track, and, if I mistake not, one that will lead to important results—new explanations of the cause of disease, new methods of prevention and treatment it may be—that will be as beneficial to mankind as they are honourable to those who pointed out the way to obtain them.

In congratulating ourselves that our countrymen abroad have taken so distinguished a part in these researches, and whilst giving all honour to our French, American, and Brazilian *confrères*, we cannot but feel satisfaction in remember-

ing how much our distinguished countryman at home has done for the elucidation of this and all other forms of helminthiasis. His researches are happily well known throughout the scientific world. I wish I could add that they were as universally appreciated as they deserve. It has been the lot of other investigators that recognition of their labour has been long postponed, as it has been in Dr. Cobbold's case; but the time will certainly come, perhaps is now at hand, when they will be appreciated at their real value. Meanwhile, he may feel the satisfaction of knowing that he has done great service to medicine, and I am glad of the opportunity of expressing, not only my own personal obligation to him for valuable information, often most kindly and freely given, but my admiration, which I know is shared by others, for the valuable information on the subject that he and others, notably Lewis, Wucherer, Bancroft, and Manson, have placed within our reach.

COMPARISON OF SMALL-POX STATISTICS, EPIDEMICS 1871 AND 1876.

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(*Read: June 12th*, 1878.)

The occurrence of an epidemic of small-pox of no mean proportions a few years after such an outbreak as that of 1871, has not unnaturally drawn the attention of the profession and the public to the continued prevalence of this disease, and it seemed to me, when requested to write a paper for this society, that a consideration of the statistics of the epidemics of 1871 and 1876 might not only be not uninteresting, but might afford valuable information. The statistics on which the present paper is based are those furnished by the reports of the medical superintendents of the various small-pox hospitals of the Metropolitan Asylum Board. For the epidemic of 1871, the figures are drawn from the reports of Drs. Collie, Gayton, Greeve, McCann, and McKellar, and for that of 1876 from the reports of Drs. Bernard, Collie, Gayton, Fox, and Waylen. I have not been able to obtain any statistics of the cases admitted into the Hampstead Hospital during the present epidemic, owing to the fact that the medical superintendent has not as yet issued any report.

Much valuable information is given in the reports referred to, but still more might have been afforded had the statistical tables been drawn up on a uniform plan; and I trust that the managers of the Asylum Board hospitals will take into consideration the propriety of having the statistical tables so drawn up.

It may be well to remember that conclusions drawn from these statistics are not absolute, and of course are only of value in so far as the admissions into those hospitals may be taken to afford a fair criterion of the nature of the prevailing epidemic.

First, with regard to the proportion of vaccinated to unvaccinated admissions in the two epidemics. Of 14,823

cases in 1871, 75 per cent. were vaccinated and 25 unvaccinated. Of 6,671 cases admitted in 1876, 74.4 per cent. were vaccinated and 25.6 per cent. unvaccinated; showing no perceptible difference in the two epidemics between the numbers of vaccinated and unvaccinated. (Table I.) It may be interesting to state that, according to Mr. Marson, the vaccinated cases from 1835-45 formed 44 per cent. of the total admissions; from 1845-55, 64 per cent.; from 1855-65, 98 per cent.; and in 1863-4, 83 and 84 per cent. respectively.

But while the proportion of total vaccinated cases to the total of unvaccinated cases remains the same in both epidemics, we find a large increase in the vaccinated admissions under fifteen years of age in 1876, as compared with 1871. Thus, in 1871 (Table III) 26 per cent. of the total number of vaccinated cases were under fifteen, as against 33 per cent. in 1876, showing an increase of 7 per cent. With regard to the number of unvaccinated cases under fifteen, we find (Table III) in 1871, 53 per cent. of the total unvaccinated admissions under that age, and in 1876, 64 per cent.—an increase of 11 per cent. This is a large increase, and one difficult to account for. One would have expected that the natural results of the Vaccination Acts would have been to increase the proportion of the vaccinated to unvaccinated cases; but one would hardly have expected that, the number of vaccinated and unvaccinated admissions remaining the same in the two epidemics, there would have been a large increase in the vaccinated cases under puberty, which is precisely that period of life in which the protective power of vaccination is supposed to be at its best.

If we analyse the statistics of the unvaccinated cases under fifteen, we find that in 1871 those under five formed 18 per cent. of the total unvaccinated, in 1876, 25.5 per cent.; between five and nine the proportion was the same in both epidemics, viz., 24.2 per cent.; and between ten and fourteen in 1871, 11.8 per cent., and in 1876, 14.7 per cent.

Now it will be observed that the most striking increase in the number of the unvaccinated cases in each quinquennial period of the epidemic of 1876, as compared with the corresponding periods in 1871, is precisely that period during which the Vaccination Act of 1871 has been in force, and the increase is no less than 7 per cent. It would be unfair to pass an opinion unfavourable to the operation of this Act, founded on the admissions into the Asylum Board Hospitals during the first five years of its operation; but it

is a fact deserving of notice that it should appear to have fallen short of what might have been expected of it.

Now with regard to the mortality in the two epidemics (Table II). Of 14,823 cases in 1871, 2,763 died, giving a mortality of 18.6 per cent. In 1876, of 6,671 cases, 1,193 died, giving a mortality of 17.8 per cent. Of the vaccinated, there died in 1871, 10.1 per cent., in 1876, 8.2 per cent.; of the unvaccinated, 45.5 per cent. in 1876, as compared with 44.6 per cent. in 1871.

According to Mr. Marson, the mortality in the vaccinated from 1836-1855, was 8.5 per cent., and in 1863-4, 10.2 per cent. I include in this all those that died of superadded disease, who Mr. Marson, for some reason or other, used to exclude from his mortality from small-pox. In the unvaccinated, from 1836-51, the mortality was 37 per cent., and from 1855-64, 34.4 per cent. It will therefore be seen that although there is a much higher mortality in the unvaccinated cases now as compared with the years from 1836 to 1864, there is only a slight increase in the death-rate of the unvaccinated in 1876, as compared with 1871. However, it is sufficient to show that small-pox in the unvaccinated is more fatal than formerly, while, on the other hand, in the vaccinated the disease is at least not more fatal. It is a curious fact that, while the mortality in the unvaccinated under fifteen was 41 per cent. in both epidemics (Table IV), over fifteen it was, in 1871, 40.5 per cent., and in 1876, 50.8 per cent. What this striking difference of nearly 10 per cent. in the death-rate of unvaccinated adults may be due to it is impossible to say.

As regards protection from small-pox, afforded by good and bad marks in those under ten years of age, the evidence afforded by the cases admitted into the Asylum Board Hospitals goes to show that good and bad marks are equally protective against attacks of small-pox, although greater immunity from death is afforded by good than bad. Of a total of 182 vaccinated cases under ten, 92 had bad and indifferent marks, of whom 5 died; 90 had good marks, of whom 2 died.

If we compare the mortality of the vaccinated cases under five in the two epidemics, we find it considerably less in 1876 than in 1871. (Table VI.) With one mark, in 1871, mortality was 26 per cent., in 1876, 10 per cent. With two marks, in 1871, 44 per cent., in 1876, 10 per cent. With three marks, 27 per cent. in 1871, and no deaths in 1876. With four marks, no deaths in either epidemic. With five marks, 33 per cent. in 1871, as against one in 1876. This

latter death is clearly exceptional, and there were only three cases. The total number of vaccinated admissions under five years of age is of course small, being 44 cases out of 4,973 vaccinated cases at all ages in 1871, and 45 cases out of 1,795 vaccinated cases in 1876. While it is disappointing to find that the number of the vaccinated attacked with small-pox under five is much more numerous in the present than in the last epidemic, it is gratifying to observe that the mortality is considerably less now than in 1871.

We have therefore seen that the proportion of vaccinated to unvaccinated admissions has been the same in both epidemics, but that while this is so there is an increase of 7 per cent. in the vaccinated admissions under fifteen in 1876, as compared with 1871, and an increase of 11 per cent. in the unvaccinated admissions under fifteen in 1876, as compared with 1871. Further, we find that the most striking increase in the unvaccinated admissions is in those under five years of age, the increase being 7.5 per cent. in 1876, as compared with the same period in 1871. We saw that the mortality in the two epidemics was nearly the same, 18.6 in 1871, and 17.8 per cent. in 1876. In the vaccinated the mortality was slightly less, 2 per cent. in 1876, while in the unvaccinated it was very nearly the same in 1871 and 1876. We have further seen that under fifteen the mortality in the unvaccinated was in both epidemics 41 per cent. Over fifteen there was an increase of 10 per cent. in 1876. Of protection afforded by good and bad marks under ten years, the evidence afforded by the statistics under review would lead one to believe that good and bad marks are equally protective against attacks of small-pox, but that greater immunity from death is afforded by good than bad marks. Finally, we saw that while the proportion of vaccinated admissions under five years had increased largely in 1876, as compared with 1871, it was satisfactory to find that the mortality was much less.

I shall now pass to the consideration of black small-pox, and conclude by some remarks on small-pox after revaccination. Of that specially fatal form of small-pox known as the black or malignant, the statistics of the two epidemics do not afford such information as may enable us to ascertain whether or not this form is of more frequent occurrence now than in 1871, but I believe it to be the opinion of those who have had large experience of both epidemics that relatively this form of disease is more frequent than in 1871.

In 1876 the proportion of black cases was, in 915 cases treated by Dr. Collie, 4.3 per cent., and in 1,594 cases treated

by Dr. Bernard about 3 per cent. Of 121 cases which occurred during the years 1871-77, of which we possess fairly definite particulars, 64 were vaccinated and 57 unvaccinated; 32 occurred in persons under puberty, and 89 in persons over that age. Of the former, 21 were unvaccinated and 11 were vaccinated. Of the latter, 36 were unvaccinated and 53 were vaccinated. Under puberty, therefore, 33 per cent. were vaccinated, and over puberty 60 per cent. Of the vaccinated under puberty, six were said to be vaccinated, but showed no marks of vaccination, four had indifferent marks, and one had one good mark. In other words, of thirty-two cases of black small-pox occurring in persons under puberty, in only five was there satisfactory evidence of vaccination. Of the vaccinated over puberty, nineteen were said to be vaccinated, but showed no marks of vaccination, six had bad marks (number not stated), four had one bad mark each, ten had two bad marks each, six had three bad, two had four bad marks each, one had one good mark, three had two good marks each, and two had four good marks each. The majority of the vaccinated, therefore, under and over puberty had bad marks, but no less than seven out of sixty-four had one or more good marks, and of these two had no less than four such marks. There is no mention, so far as I am aware, of this form of small-pox having occurred in a revaccinated person.

That the black small-pox occurs in many vaccinated persons we have seen to be a fact, and for this reason it has been concluded that vaccination confers no protection against black small-pox. It would in my opinion be as reasonable to say that vaccination did not protect generally against the mild or the severe forms of small-pox, because in a certain proportion of cases it fails to do so. Good vaccination protects absolutely against no form of small-pox, but the number of black cases in those with good vaccination marks is few. Those who hold the opinion that vaccination confers no protection against black small-pox, appear to me to be in error, first, because the majority of the malignant cases occur in persons over puberty, when the protection originally conferred by vaccination fails, not only as against black, but as against all other forms of small-pox; second, because the majority occur in badly vaccinated persons; and third, because black small-pox in a vaccinated person under puberty is extremely rare, and, in a revaccinated person, unknown.

On the subject of the protection afforded against small-pox by successful revaccination about puberty, the state-

ments contained in the reports of the medical officers of the asylum district hospitals are contradictory. On the one hand we find that they are unanimous in saying that none of the staff who have been revaccinated successfully have contracted small-pox. But, on the other hand, Dr. Collie and Dr. Gayton give instances of small-pox after revaccination in patients. Dr. Gayton says, "Out of the whole number, thirteen (of cases of small-pox after revaccination), which have come under my observation, in five only could satisfactory evidence be discovered of successful revaccination." It would have been interesting to have had some information as to the age of the patients, and the age at which revaccination was performed. Dr. Collie gives particulars of six cases of small-pox after revaccination. In three, marks of revaccination were present, and one of these died. Dr. Collie's note on this case is "M. F., aged 18, said to have been vaccinated in infancy, and revaccinated aged 12. There are two scars on the left arm, which, if due to vaccination, are bad." Of the others, one had no mark, but the patient stated that her arm was "bad" after the operation, and the remaining two were said to have been revaccinated, but had no marks of revaccination. In my own experience at the Deptford Hospital I have seen three cases of small-pox after revaccination out of 204 cases admitted since the hospital was opened in April last. One patient, aged 19, who has three indifferent primary marks, possesses two marks of revaccination, performed when aged 16. Another, a female aged 20, with two indifferent marks of primary vaccination, was revaccinated when aged 16, and possesses three fair-sized marks of revaccination. In both cases the attack of small-pox was very mild. Another patient, aged 18, with two bad primary marks, was revaccinated when aged 11, but possesses no marks of revaccination; has had a mild attack of small-pox. Of twenty-two recorded cases then we find that one died, giving a mortality of 4.5 per cent., which is just about the mortality of the vaccinated cases under puberty. It would be clearly incorrect therefore, in the face of these facts, to say that revaccination is preventive absolutely of small-pox. For myself, I am inclined to believe that small-pox after successful revaccination is not infrequent; but it is to be observed that severe or fatal cases are rare, and that in the latter revaccination has probably been performed, as in the fatal case recorded by Dr. Collie, under puberty.

TABLE I.*

Showing the percentage of the vaccinated to the unvaccinated admissions in the two epidemics.

	1871.		1876.	
	Cases.	Percentage.	Cases.	Percentage.
Vaccinated	11,174	... 75.0	4,978	... 74.4
Unvaccinated	3,649	... 25.0	1,693	... 25.6
Total	14,823		6,671	

NOTE.—The numbers for 1871 are taken from the reports of the Medical Superintendents of the Hampstead, Homerton, and Stockwell Hospitals; for 1876, from the reports of the Medical Superintendents of the Homerton Fever and Small-pox Hospitals, Stockwell Small-pox Hospital, and Deptford and Fulham Hospitals.

TABLE II.

Showing the mortality of the vaccinated and unvaccinated in the epidemics of 1871 and 1876.

	1871.		Mort.	1876.		Mort.
	Cases.	Deaths.	per cent.	Cases.	Deaths.	per cent.
Vaccinated	11,174	1,135	10.1	4,978	422	8.2
Unvaccinated ...	3,649	1,628	44.6	1,693	771	45.5
Totals	14,823	2,763	18·6	6,671	1,193	17 8

NOTE.—Table II from same sources as Table I.

TABLE III.

Showing ratio of vaccinated to unvaccinated under and over fifteen years of age in 1871 and 1876.

(α) Under 15.

	Vaccinated.	Unvaccinated.	Total.
1871	1,111	922	2,033
1876	684	631	1,315

(β) Over 15.

	Vaccinated.	Unvaccinated.	Total.
1871	3,092	809	3,901
1876	1,353	359	1,712

	Vaccinated.	Unvaccinated.
Totals for 1871	4,203	1,731
,, 1876	2,037	990

Percentage of vaccinated under fifteen to the total vaccinated was, therefore, in 1871, 26, and in 1876, 33. Percentage of unvaccinated under fifteen to the total was, therefore, in 1871, 53, and in 1876, 64. (Marson, 1836–51—36 per cent. unvaccinated under fifteen.)

NOTE.—For 1871, figures taken from Stockwell Small-pox Hospital and Homerton Fever and Small-pox Hospital Reports. For 1876, figures taken from Homerton Small-pox and Fever Hospitals Reports.

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* In the following tables the unvaccinated include all those who had no marks of vaccination.

TABLE IV.

Showing the mortality in vaccinated and unvaccinated admissions under and over fifteen years of age in 1871 and 1876.

(*a*) Under 15.

	Vaccinated. Cases.	Deaths.	Mort. p. cent.	Unvaccinated. Cases.	Deaths.	Mort. p. cent.
1871	1,111	60	5.4	922	376	41.0
1876	684	33	4.8	631	259	41.0

(*β*) Over 15.

	Vaccinated. Cases.	Deaths.	Mort. p. cent.	Unvaccinated. Cases.	Deaths.	Mort. p. cent.
1871	3,092	340	11.0	809	328	40 5
1876	1,353	130	10.0	359	182	50.8

NOTE.—Numbers from same sources as Table III.

TABLE V.

Showing the number of cases and number of deaths in the unvaccinated admissions in periods of five years up to fifteen (1871, 1876).

	1871. Cases.	Deaths.	Mort. p. cent.	1876. Cases.	Deaths.	Mort. p. cent.
Under 5 yrs.	658	433	65.8	177	109	61.5
From 5–9	372	154	41.6	132	57	43.1
„ 10–14	272	75	27.6	46	12	26.0

NOTE.—1871: Under nine years numbers are taken from reports of Drs. Collie, Gayton, Grieve, McCann, and McKellar; total unvaccinated cases admitted, 3,649. From ten to fourteen from reports of Drs. Collie, Gayton, McCann, and McKellar; total unvaccinated admitted, 2,321.—1876: Under nine, numbers taken from reports of Drs. Bernard and Collie, total unvaccinated, 694. From ten to fourteen, numbers taken from Dr. Collie's report, total unvaccinated admissions, 312.

From the above table and note it is evident that the percentage of unvaccinated admissions in the three quinquennial periods up to fifteen, to the total unvaccinated admissions, will be, in the two epidemics, as follows:—1871: Under five, 18.0; from five to nine, 24.2; ten to fourteen, 11.8 per cent. 1876: Under five, 25.5; from five to nine, 17.5; ten to fourteen, 14.7 per cent.

TABLE VI.

Showing number of cases and number of deaths in vaccinated persons under five years of age, with vaccination marks, irrespective of quality.

	One mark.			Two marks.			Three marks.			Four marks.			Five or more marks.		
	Cases.	Deaths.	Mort. per cent.	Cases.	Deaths.	Mort. per cent.	Cases.	Deaths.	Mort. per cent.	Cases.	Deaths.	Mort. per cent.	Cases.	Deaths.	Mort. per cent.
1871	15	4	26.6	9	4	44.4	11	3	27.2	6	—	—	3	1	33.3
1876	10	1	10.0	9	1	10 0	8	—	—	14	—	—	4	—	—

The figures in 1871 are Dr. Grieve's, in 1876 Drs. Bernard's and Collie's.

TABLE VII.

Showing the protection afforded by good, bad, and indifferent marks in persons under ten years of age. 1876. Total vaccinated cases 1,795.

GOOD MARKS.

One.		Two.		Three.		Four.		Five or more.	
Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
13	1	22	—	20	—	28	1	7	—

Total cases with good marks, 90; died, 2.

BAD MARKS.						INDIFFERENT MARKS.					
One.		Two.		Three.		One.		Two.		Three.	
Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
14	2	6	2	9	—	12	—	15	1	36	—

Total cases with bad and indifferent marks, 92; died, 5.

NOTE.—This table is drawn up from reports of Drs. Collie and Bernard, 1876-77.

TABLE VIII.

MALIGNANT OR BLACK SMALL-POX.

	Under 15 years.	Over 15.
Unvaccinated (confessedly)	17	28
No evidence of vaccination	4	8
Said to be vaccinated (no marks)	6	19
With bad marks (number unknown)	—	6
„ one bad mark	—	4
„ two „	—	10
„ three „	—	6
„ four „	—	2
„ indifferent marks	4	—
„ one good mark	1	1
„ two good marks	—	3
„ three good marks	—	—
„ four good marks	—	2
Totals	32	89
	121	

NOTE.—This table is drawn up from reports of Drs. Collie and Gayton for 1876 and 1877.

DETAILS OF EXPERIMENTS ON THE VACCINATION AND SUBSEQUENT REMOVAL OF SUPERNUMERARY FINGERS,

APPARENTLY SHOWING THE IMPERFECT PROTECTION AFFORDED BY VACCINATION WHEN THE VESICLES ARE REMOVED BEFORE THEIR FULL MATURITY.

By ROBERT CORY, M.A., M.D. Cantab.,
Assistant Obstetric Physician to St. Thomas's Hospital.

(*Read: June 4th*, 1879.)

In relating the details of the following experiments, I fear I am going to be a little tedious. The cases I have met with suitable for experiment have only been four in number, so that it becomes necessary to record carefully their results, together with the conditions that may have modified them. I will proceed at once to the details of the experiments, and then offer a few remarks upon what I take to be their significance.

Experiment I.—Eliza Page was born in Southwark with a supernumerary thumb. This additional thumb articulated with the metacarpal bone, and hence had a broad attachment with the hand. On the 21st of April, the child then being seven weeks old and in good health, I vaccinated it on the tip of the thumb with lymph taken in a tube on the 11th of April. On the 23rd, 49 hours after vaccination, there was slight redness about the points of inoculation. On the 24th, 60 hours after vaccination, the redness was more marked, but as yet there was no appearance of a vesicle. On the 25th, 85 hours after vaccination, the inoculated places were distinctly vesicular. On the 26th, 108 hours after vaccination, the vesicle had increased in size, and an areola was present, but this did not extend to the hand. It was at this stage of the development of the vesicle that the thumb was amputated by Mr. Mason.

For the next three days an inflammatory blush was present over the hand. This subsided on the tenth day from vaccination, and the wound after this healed rapidly.

On the 30th of May 1877, 39 days after the vaccination on the thumb, I vaccinated the child in five places on the left arm directly from another child. This vaccination did not take. I therefore again vaccinated the child on the 6th of June, which was also done directly from another child, with no better result.

As my experience in arm to arm vaccination since the beginning of 1876 has been four failures in about 3,000 cases, and I have never failed to vaccinate on a second attempt, I think I was justified in assuming that this child received full protection from the first vaccination.

Experiment II.—After this case, I had no further opportunity of experimenting until Jan. 15th, 1879, when Dr. McKellar kindly sent me a child unvaccinated and having a supernumerary thumb, similar to the one just mentioned. The child, Frank Williams, resided at 59, Lothian Road, Camberwell. He was vaccinated on his supernumerary thumb on the 15th of Jan. 1879, being then five weeks old, directly from another child. On the 18th, there was no evidence of the vaccination having taken, and the thumb was accordingly re-vaccinated on this date from a tube taken on the 15th of Jan. On the 20th, when I again saw the child, I felt certain the first attempt to vaccinate the thumb had failed, but I was uncertain about the result of the second. Eczema existed behind one ear, which the mother stated first appeared on the 18th, after my visit. On the 22nd, I found neither place had taken, and as the eczema was worse, I postponed further attempts to vaccinate the thumb until this had cleared off.

On the 15th of Feb. the eczema had entirely disappeared, and I therefore again vaccinated the thumb with lymph taken in a tube on the 12th of Feb., making three insertions with a grooved needle, as remote as possible from the hand. On the 19th of Feb., 96 hours after the vaccination, the three places had all taken, and were vesicular. I accordingly removed the thumb, taking precautions not to injure the vesicle, for otherwise the stump might have been inoculated with the lymph contained in it. On the 22nd, the wound was doing well, an area of inflammation was present on the hand, which was not present, as I was informed by the mother, on the preceding day. On the 25th, the sutures were removed. The area of inflammation was then declining.

On March 5th, the wound was healed. The mother was then told to bring the child up in a month to be vaccinated at St. Thomas's Hospital, but as she had not done so by the 18th of April, I called to see her, and found that

she had had the child vaccinated on the 8th of April by Mr. Pinder of Camberwell. On looking at the arm on this the eleventh day after vaccination, I found four small brown scabs quite dry and almost ready to fall. The areola, which I was informed had been very extensive, had quite subsided. I also learnt that the redness began to come very soon after the vaccination, and that when she took it to the station on the 15th, it extended to the elbow. In this case the early and extensive areola, its early subsidence, and the rapid drying of the scabs, all indicated the rapid progress of the vaccination.

Experiment III.—The third case I met with was one kindly sent to me by Mr. Chilton. This was a child named Henry Pike, born on the 29th of December 1878, at 8, Greys Place, George Street, Lambeth. He had, like the other cases, a supernumerary thumb.

On the 22nd of January I vaccinated the thumb directly from the arm of another child. On the 25th of January, seventy-two hours after vaccination, there were distinct evidences of the vaccination having taken. I therefore removed the thumb. On the 26th there was no appearance of inflammation about the hand. On the 27th there was slight inflammation at the lower angle of wound. On the 28th the inflammation was a little more extended. On the 29th the inflammation was still more extended. On the 30th the extent of the inflammation was about the same as on the 29th. The union of the wound was complete, and the sutures therefore removed. From this date the redness declined.

On the 19th of March I vaccinated the child in five places on the arm directly from another child. On the 26th all five places had taken. The areola, which commenced early on the preceding day, was apparently at its height on this the eighth day. On the 28th, which was the tenth day after vaccination, the areola was subsiding. There was an enlarged gland in the corresponding axilla.

On April the 3rd two of the scabs fell, and the other three fell on the following day, which was the seventeenth day from the vaccination. In this case the course of the vaccination was rapid, though not so rapid as in Experiment II, in which the finger was removed on the fifth day.

Experiment IV.—This was a child named Thomas Moreton, born at 15, Upper Marsh in December 1878, who had a supernumerary little finger which did not articulate with the bone of the hand, but was connected to it merely by a slight pedicle.

I vaccinated this finger bud on the 14th of February from a tube taken on the 12th of February. On the 18th of February, ninety-seven hours after vaccination, I removed the finger by tying a silk ligature round its pedicle, and cutting it off beyond the ligature with a pair of scissors. There was no sign of any inflammation until the 21st, when there appeared on the hand a little redness surrounding the ligature. This increased to about three-fourths of an inch radius on the 22nd, and began to decline on the 23rd. The ligature came off on the last mentioned day. There was no axillary enlargement or tenderness.

On the 2nd of April I vaccinated this child on the arm directly from another. On the 9th of April all five places had taken, and the areola was then at its height, and rather extensive. On the tenth day the areola had declined. The scabs fell early, but the exact dates I was unable to learn.

In considering these experiments there are two results which appear striking. The first is that in all the cases, on the eighth day after the first vaccination, an area of inflammation was present on the hand from which the vaccinated finger had been removed. This was most remarkable in the fourth experiment, for in this case the finger was attached by a slender pedicle, which was ligatured, and the extent to which the inflammation reached on the ninth day was greater than might have been expected merely from the irritation of the ligature. If this inflammation was not solely due to the irritation of the wounds it must have been occasioned by the same conditions which lead to the development of the areola after ordinary vaccination. I do not, however, wish to lay much stress upon this point.

The second result is the most important, for it would appear from the three last cases that although protection against a second vaccination was not given by the first vesicles, which were removed, yet these vesicles did produce a certain effect, which caused the second vaccination to run rapid courses.

It is well known from Bryce's list that when a second vaccination is performed on the fifth day after the primary, the vesicles which result run a rapid course, so that they overtake the primary vesicles, and both mature together. The same thing happens when the second vaccination is done later than the fifth day, as the following case proves.

On the 2nd of March 1876, I vaccinated a child, Edward Pillar, in four places on the right arm, from a tube taken on the 1st March 1876. On the 8th of March, the seventh

day afterwards, I vaccinated another place on the left arm, and I continued to vaccinate a place daily on this arm until the 14th of March. The places vaccinated on the seventh, eighth, and ninth days after the primary vaccination took, the one, however, done on the last-mentioned day scarcely became vesicular, although it had on the 13th of March a slight areola round it. The places done on the tenth, eleventh, and twelfth days did not take, and no areola appeared round them. On the 17th of March all the scabs on both arms were desiccated. The whole course of the vaccination was thus seen not to have been prolonged by the later vaccinations, for the vesicles thereby formed rapidly developed, and overtook the primary ones. The ninth day after the primary vaccination was in this case the latest day, any result could be obtained by a fresh insertion of lymph, and this is precisely the result which Trosseau had observed in his experiments, mentioned in page 121, vol. i, of his work on *Clinical Medicine*, third French edition.

As the vaccine vesicles usually increase in size up to the ninth day, but not afterwards, we see that the last day we can vaccinate with effect is also the last day the vesicles increase in size; and this is what we might expect, for the vaccine vesicles will increase in size until the skin has undergone that unknown modification which renders it incapable of being further affected with the vaccine virus. When this has taken place, the vesicles cannot spread.

Now, suppose in one of the above experiments we had vaccinated the child in another portion of the skin on the same day the finger was amputated. What would have been the result? I am inclined to think this second vaccination would have run a rapid course, just as we have seen it would have done if the primary vesicle had not been interfered with. In other words, the amputation of the finger would not have prolonged the time ordinarily occupied between the first insertion of the virus and the desiccation of the scabs.

From these considerations it seems probable that the modification, whatever it may be, which renders the system incapable of further infection with the vaccine virus, is a progressive one, commencing at the time, or shortly after, the first insertion of the virus, and goes on, hand in hand, with development of the vesicles.

As I take it, what the above experiments seem to show is, that this progressive modification is arrested when the vesicles are removed; and that, when vaccination is again performed, the modification, already partially accomplished,

proceeds to completion from the point at which it had arrived when arrested.

Another matter here suggests itself for consideration, and it is the likeness which the rapidly developing vesicles of the second vaccination in three of the above experiments presented to the vesicles in ordinary cases of secondary vaccination. The great characteristic of a secondary vaccination is, that it runs a rapid course, and has an early areola. As a rule, the shorter the time that has elapsed between the primary and the secondary vaccination, the more rapid is the course, and the slighter the results of the latter. A probable explanation of this likeness is found in this consideration. If the modification wrought in the system during the course of a primary vaccination be progressive, as we know the return from a state of perfect protection to the original condition is progressive, we should expect that a vaccination performed, either when the modification had been arrested halfway, or when the modification had half passed off, would give rise to the same phenomena.

I think we have now seen reason for believing, that if in the course of a vaccination the vesicles be removed before their full maturity, the protection afforded by the vaccination will only be partial, and that the time requisite for the system to reacquire its original susceptibility to the virus will be shorter than if the vesicles had not been interfered with; and this leads us to the question—may not those conditions of the skin, such as eczema, which are known to influence the development of the vesicles, be also effective in arresting the modification of the system? If this be so, we would then have an explanation for some of those cases in which small-pox has early followed vaccination, as well as a reason why, with slight scars, the protection against small-pox is not so great as when the scars are well marked, for a slight scar may very well be taken to indicate that the vesicle which gave rise to it did not, from some cause or other, attain its full development.

I should be exceeding the limits of this paper if I were to enter into other questions suggested by the above experiments; besides, I cannot but feel that I have already built too much upon what, from the small number of the cases, is but a poor foundation. I have brought them forward, because supernumerary fingers are not met with every day, and I thought others, having the opportunity, might care to experiment.

THE ETIOLOGY OF SUMMER DIARRHŒA.

By WILLIAM JOHNSTON, M.D., F.G.S.,

Assistant Officer of Health to the Borough of Leicester.

(*Read: January 1st*, 1879.)

Of the whole class of zymotic diseases, diarrhœa is the one which demands the greatest effort to be put forth towards an investigation of its causes. Why this disease should possess such prior claim upon our attention depends upon the following reasons.

1st. Amongst urban populations, if an average be taken for a number of years of the deaths which each zymotic disease has induced, it will be found that the number accredited to diarrhœa considerably exceeds that of any other of this class of maladies.

2nd. The majority of the victims are children.

3rd. The wide difference of opinion that is observed to exist amongst health officers with respect to its mode of causation, and which in turn gives rise to hesitancy and apathy on the part of the sanitary authorities throughout the country in their adoption of measures for the mitigation of the disease.

4th. The disease arises from causes pre-eminently under control.

Leicester is one of the English towns which has for some years past enjoyed unenviable notoriety for a high diarrhœa death-rate. Early in the summer of last year, the Sanitary Committee of this town, with a view to lessen, if possible, the fatality from this disease, determined upon supplying medicine *gratis* to the artisan and poorer classes. In the adoption of this measure the Committee were chiefly influenced by the good accounts received from Manchester, where a similar line of action was resorted to. On the 10th July I directed that the addresses and ages of all the persons for whom medicine was given should be entered, so that I might ascertain the ages of those affected, and the districts where the disease was most prevalent. The following is an analy-

sis of the entries from the 10th July to Sept. 30th. Total number of cases affected, 3,318.

1st. Of these 3,318 cases, 1,590 were children and 1,728 adults, thus showing that the disease was more prevalent amongst adults than children.

2nd. The applications from houses where more than one was affected with the complaint were 481 in number, viz.:

Houses having 7 persons suffering from the disease, 1
" " 6 " " " " 3
" " 5 " " " " 8
" " 4 " " " " 22
" " 2 or 3 " " " " 220
Houses having the parents affected with the children, 227.

The history of these multiple cases was, almost without exception, that first one became affected with the disease, and that the others subsequently suffered from it.

During July and up to the 9th August, I was engaged in rather lengthened microscopic examinations of the bowel discharges from infants and adults, and during this time I acquired the disease no less than five different times. Each attack readily yielded to strong doses of quinine and dilute sulphuric acid; but after a lapse of some days, and upon resuming my examinations, a fresh attack nearly always followed in from twenty-four to forty-eight hours. These personal experiences, especially when taken in conjunction with the facts mentioned above, under paragraphs 2 and 3, have convinced me that summer diarrhœa is *contagious*, and that the chief vehicles of the poison in the above instances were the ejecta from the bowels of previously infected persons.

The characters of the evacuations in infantile cases are peculiar. As they issue from the bowel they are, in the vast majority of instances, alkaline in reaction, most offensive in odour, and often ammoniacal. They vary exceedingly in appearance, some being deeply tinged with bile, while others are of a pale clay colour, or straw colour, or greyish, and these various hues are all more or less disguised and modified by variable quantities of undigested food. Upon standing, the evacuations separate into two layers, the upper being shallow and watery, and the lower formed of a sediment of the consistence of moderately thick starch, and composed principally of articles of nourishment in an undigested state. Now, if a small quantity of the upper layer, or a little of the watery portion of a stool that is just evacuated, be collected by means of an ordinary clean lymph

tube and submitted to microscopic examination, there will be observed: (1) Particles of undigested food; (2) Fat globules in greater or less profusion, and these invariably accompanied with (3) innumerable bacteria. These bacteria are of two genera, viz., (*a*) minute glistening single spherules of micrococcus in active jostling movement, and (*b*) numerous rods of bacterium termo, some in a resting condition, but the great majority in rapid swimming and tumbling motion. The staves vary somewhat in length, and along the centre of some there may be detected an extremely faint line.

These organisms are the active agents in causing putrefaction in animal nitrogenous substances; and in any putrescible liquid containing albuminoid organic matter, the establishment of putrefaction depends upon the development and multiplication of these infinitely small organised living beings.

In the fresh liquid ejecta of adults, I observed, in addition to the forms mentioned above, considerable numbers of staves, some bent, but the greater number straight, and much longer than the ordinary bacterium termo; these moved actively in a sliding and slightly undulating manner, and the locomotive power of some was so great as to enable them to cross the field of the microscope with rapidity. In samples of adult stools kept exposed to the air at a constant temperature of about 70° Fahr., I observed numerous chains of micrococci in the course of twenty hours. The appearance of this new species may, in my opinion, be accounted for by an admixture of urine with the fæces taking place during the action of the bowels, as similar chains are always found in urine undergoing putrid decomposition. All recent specimens of adult stools also contained an abundance of crystals of ammoniaco-magnesian phosphate, some of which were of large size. I have never detected these crystals in the fresh evacuations of infants. I may mention, before leaving this part of the subject, that I have never once found the stools of any of forty-nine patients (aged, adult, and infantile) affected with the disease to be free from these organisms; but, on the contrary, they invariably contained bacterium termo and micrococci in great numbers. The youngest patient whose stools I examined was only three weeks old, and the age of the oldest patient was seventy-six years. I have never been able to detect bacteria in the liquid motions of infants during health, and amongst my patients I observed that the gradual disappearance of the bacteria from the stools was in great measure commensurate with their recovery.

In the vomit of seventeen breast-fed infants affected with "summer" diarrhœa, I invariably found rods of bacterium termo, and highly refractive micrococci. The relative proportions of these bodies, as observed in the bowel discharges, seemed to be reversed in those from the stomach, as here the glistening micrococci appeared, amongst numerous oil globules, in greater number than the rods; most of the latter were also motionless, and the numbers of both forms were very much less than in the evacuations.

Theories of Causation.—Before proceeding to discuss the theories at present advanced to explain the causation of the disease amongst urban populations, I wish to draw brief attention to the facts recorded upon this paper. They embody the result of a house-to-house visitation I made during last November to all dwellings in which deaths occurred from diarrhœa during the epidemic of 1878.

I may say that the epidemic mortality began to be felt at a much earlier date than in 1877. Three deaths were recorded from the disease on June 27th, and from this date until the 25th August, no intermission took place in the daily return of diarrhœal deaths. The total deaths recorded from 27th June to 30th September were 276. Only nine of these were adults, the remaining 267 were infants and children under three years.

The first theory I shall notice, that is advanced by many as the chief cause of the disease, is *Artificial Feeding*. We all know that this habit is very prevalent amongst mothers in town populations; and we are all equally aware of the fact that its adoption tends very materially to enfeeble the infantile organisation, and so render it more unable to withstand any disease-inducing influence surrounding it. No member of the profession deprecates more strongly than I do the adoption by mothers of this unnatural custom, and I am fully satisfied that much infantile mortality might be averted by its discontinuance. At the same time, I think that the evils ascribed to artificial feeding are, just at present, very much over estimated; and with respect to its being the principal cause of the diarrhœal fatality of our large towns, I am of opinion that the facts to be gleaned from last year's epidemic by no means lend support to such an assumption, for we find that of 238 deaths in Leicester, about which full particulars could be obtained, no fewer than 165, or 69.3 per cent. of them were breast-fed, etc.

The seventeen infantile cases which occurred amongst my own patients, and to which I have previously referred

as having bacteria in their vomit, were all breast-fed, and had never from their birth received artificial food of any description whatever. All, however, suffered from a grave form of the disease, and three out of the seventeen succumbed to the attack. Again, the development of last year's epidemic was sudden and contemporaneous over certain districts of Leicester, and the disease affected all ages simultaneously. This affords strong proof that the cause in operation was rapidly diffused through a medium common to these inhabited areas. Nothing like a limitation of the disease to the infantile portion of the population was observed. The mortality arising from the disease was, as we have seen, almost entirely confined to infants; but this is not to be wondered at, when we take into consideration the delicate constitutions of the victims.

A few facts relative to the diarrhœal mortality sustained in other towns during the summer quarter may not here be out of place.

The industries of Oldham and Bradford employ a greater percentage of female labour than most other manufacturing towns, and certainly much more than Leicester. Notwithstanding this, however, Oldham suffered the least fatality from the epidemic of any of the twenty large towns, and Bradford, with a surplus population of 64,000 over that of Leicester, sustained only 36 per cent., the actual mortality of the latter town.

A similar disparity is observed in the diarrhœal death-rates of towns engaged in similar branches of industry, and employing much the same proportion of female operatives; *i. e.*, Halifax suffered a remarkable immunity from the disease, its diarrhœal rate for the summer quarter being only 1.3 per 1,000; but, for the same period, the rate in Leeds was 6.9; in Preston 7.8, and no less than 8.1 in St. Helen's. From my own personal knowledge, artificial feeding is carried on to an enormous extent in Halifax and Oldham; and if the disease mainly depended upon this custom, one would be prepared to find both towns suffering from heavy yearly inflictions of diarrhœa, instead of which both places enjoy a remarkable immunity from the malady.

To take another striking example: Bristol and Hull are both sea-port towns, and their employment of female labour bears but a small percentage to their populations. Bristol, however, was noted for its low diarrhœal death-rate, whilst Hull, during the same quarter, experienced a higher fatality from the disease than any other English town. With such conclusive evidence before me, I cannot accept

artificial feeding, *per se*, as a cause essential in the production of the disease.

Other theories advanced as causes of the disease are: impure water, maternal neglect, want of cleanliness, and ventilation.

We find, as regards the water-supply to the houses in Leicester where deaths occurred, that 193 or 81.1 per cent. of the houses were supplied with the Company's water, and only 45 with pump-water (18.9 per cent.). The Company's water is plentiful, and the supply constant. Several analyses of the water during the summer showed that it was of excellent quality. The disease, in Leicester, is therefore not dependent upon bad water-supply. The closet accommodation to the houses was also good; only eleven privies were met with, the remaining 227 habitations having either pails or W.C.'s provided. The removal of ash-bin refuse is most efficiently carried out. These figures afford conclusive evidence that the disease, as far as Leicester is concerned, is neither dependent upon filthy conditions of its surface, nor due to any want of the ordinary sanitary requirements for external cleanliness.

A somewhat striking fact was also elicited during my enquiries into the particulars attending last year's epidemic. In the courts throughout Leicester, and amongst their occupants, more filthy tenements, more squalor, less facilities for house ventilation, greater maternal slovenliness, and deeper degrees of poverty, are found to exist than in any other district or section of its general population. The mortality, however, occasioned by the disease amongst the progeny of these people, was not found to exceed that experienced by the inhabitants of more open districts of the town; but, on the other hand, a somewhat remarkable fatality took place in some wide, open streets, newly built and situate upon the extreme margin of the town.

All the evidence I have now laid before you in discussing the generally accepted theories of the causation of diarrhœa tends to show, in my opinion, that these ascribed causes fail to account for the wide variations in the mortality experienced each summer quarter in English towns.

Before entering upon this subject, I may state that I consider atmospheric conditions act only as *excitants* of the disease, and this only applies to localities where other conditions are met with that act powerfully as *predisposing* causes, for I feel assured that if we could discover any par-

ticular town or district whose sanitary machinery was in perfect working order the vicissitudes of climate would there be found to be perfectly harmless in the production of the disease.

I have arrived at the following conclusions with respect to the weather conditions that seem to influence diarrhœa mortality, after a careful tabulation of the meteorological conditions and diarrhœa mortality experienced during the summer months of each year since 1866. The prevalence of the disease appears to be governed by three weather conditions, viz., temperature, atmospheric humidity, and rainfall, and these follow in the order of their importance. The combination of these that seems most potent for evil is high degrees of heat, great atmospheric dryness, with little or no rainfall. This combination was experienced throughout July of 1868, and the diarrhœa mortality was greater than has ever since been recorded. These conditions of weather, it will be observed, exactly coincide with those pre-eminently favourable to rapid aqueous evaporation. Very high temperatures prevailed during the months of May and June of the same year. The combination most unfavourable for the disease seems to be prevailing low mean temperatures ranging from 37° Fahr. to 58.5° Fahr., in conjunction with high degrees of atmospheric saturation, and more or less continuous rainfall. The least mortality (86 cases) was experienced in Leicester during a prevalence of these conditions in the summer quarter of 1866. The latter weather conditions are most unfavourable to rapid aqueous evaporation.

From the charts of the previous twelve years, I think this general law may be gathered, that a continuance for a week or ten days of a mean daily temperature of 59° or over, in conjunction with a scarcity of rainfall and low degrees of humidity, and occurring during any period from the twenty-fifth to the thirty-seventh week, is sufficient either to provoke the disease or increase its prevalence. A hot June month invariably hastens its appearance.

Before entering upon the mode of causation of the disease, it is essential that I should briefly draw attention to the change of temperature invariably induced in the subsoil by the meteorological conditions experienced during the spring and summer quarters of the year. The sun is the principal source which imparts to the subsoil of our towns the heat which it possesses. From the vernal equinox, March 21st, until about June 22nd, the days gradually increase in length, and the heat which the soil receives from solar radiation during the days is, speaking generally,

greater than it parts with during the nights. The immediate result is a gradual rise during the spring quarter in the temperature of the subsoil and the sewers embedded therein. The temperature in sewers, when these are not sufficiently ventilated, and under ordinary conditions of weather, will be found to have risen from 52° or 54° Fahr. in March, to 59° or 62° by the end of June; and if hot, dry weather has prevailed during this latter month, much higher sewer temperatures may be registered at its close. A decided cooling of the sewers and subsoil of towns, as indicated by temperatures below 56°, will not be observed to take place, under ordinary conditions, until about Sept. 25th, *i.e.*, until the period just before the daily receipts of heat from solar radiation begin to fall short of the nightly expenditures. The highest sewer temperature I have ever observed was 69.5° Fahr.; this was taken on the 19th July of the present year (1878), when the outer air temperature was 83.5°.

Now, when temperatures of 56° Fahr. and over are once established in sewers, putrid fermentation sets in, and the process goes on with a rapidity that is directly proportionate to the increase of temperature attained in the sewer. Sewers in this heated state afford the very conditions (heat, air, and moisture) for the rapid putrefaction of all the putrescible liquids and animal excreta that may be retained in them, for the air of a sewer, if not sufficiently ventilated, is always at the point of *complete saturation.*

Origin of the disease.—My observations, during the last eighteen months, upon the microscopic organisms contained in ordinary air and that of sewers, are much too numerous to mention in this already too lengthy paper; I will, therefore, only draw attention briefly to what I have observed, and the conclusions I have drawn therefrom.

1. During the summer months, the liquid portion of sewage derived from sewers of deposit will be found, upon microscopic examination, to contain great numbers of living forms, including (*a*) several genera of bacteria, such as micrococcus, bacterium termo (free and in the zooglœa form), bacillus, and vibrio; (*b*) rapidly moving infusoria, flagellate and ciliated, belonging to several kinds of common protozoa, as monadina, euglenia, alyscum, trichodina, paramecium, kerona, etc.; and (*c*) microscopic hirudinea. The above living forms are always accompanied with variable quantities of the spores, spore-cases, and mycelium of fungi.

2. The *moist air* in all ill-ventilated and non-cleansing sewers, when the temperature of the latter is above 57 deg.

Fahr., contains bacteria of the genera micrococcus and bacterium termo, the numbers present being in direct ratio to the increase of temperature from 57 deg. to 69.5 deg. (the highest temperature observed).

3. The same organisms are to be found in the confined air of cesspools, and here their numbers increase with the atmospheric temperature.

4. The trapping water of all gulley-grates and stench-traps, when connected with sewers of deposit, contains great numbers of both kinds of bacteria, derived in great measure *from the air within.*

The air of an insufficiently ventilated system of sewers undergoes a daily range of temperature varying from 3 deg. to 12 deg. Fahr. The greatest daily fluctuations take place in the air of main sewers; in these the lowest temperatures occur between 3 and 4 A.M., and the highest between 5 and 6 P.M. The temperature of the air in the sewers of a manufacturing town is never, at any given moment, uniform throughout the system, for, owing to the frequent receipts of heated water from factories and other sources, the temperature in the low-lying main culverts will be found throughout the day to be from 5 deg. to 9 deg. Fahr. higher than that in the subsidiary sewers radiating from such culverts. An exactly opposite relationship with respect to these sewer temperatures will be found to exist during the greater part of the night, for then the temperature of the air in the main culverts is lower than in their tributary sewers.

Too great importance cannot, in my opinion, be attached to this *increasing* variation in the temperature of the air of town sewers. Owing to this variation, ascending aërial currents are established during the day, *irrespective of the low specific gravities of some of the component gases,* and if from defective construction or insufficient fall any section of the main culverts retains excretal matters and becomes silted, the specific contaminations which such matters contain are thus carried into the remotest branch sewers, though these latter may extend to considerable distances, and occupy much higher levels. Stagnation and deposit occurring in a main sewer soon impart a common character to the air of all its confluents.

5. During hot weather, and especially when conjoined with great atmospheric dryness, these organisms are given off from all liquids containing them, and are carried into the air in very large numbers by the ascensional force of evaporation. This fact can be easily demonstrated by

covering any vessel containing a solution abounding in bacteria with a glass plate. The under surface of the plate becomes coated with watery vapour, which can be collected into drops by the addition of ether, and on subsequent examination discloses the presence of bacteria. These bacteria, as has been before stated, are the active agents in causing putrefaction in putrescible liquids and substances of animal organic origin, and their appearance in the air of sewers and close cesspools is a sure indication that rapid putrefactive changes are going on in the excrementitious matters that may be retained in the former or collected in the latter.

6. In badly sewered districts, milk will be found to be speedily infected with the above ferments; their presence may also be frequently recognised in the stale food of infants if due care be employed in their collection.

7. The juices that exude from and adhere to the overripe fruit, exposed in shops or hawked about in such districts, will be found, with few exceptions, to contain numbers of bacteria (rods and spherules) moving about amongst the cells of saccharomycis exiguus (Rees), the ordinary variety of fruit ferment.

8. The atmosphere of these districts during hot summer weather when filtered is always found to yield fungal elements, ranging in character from micrococcus to mycelial filaments. I have never detected the presence of bacteria in the air of the same localities during any day in April or May.

9. For the last two years the commencement of "summer" diarrhœa in Leicester has been contemporaneous with the appearance in large numbers of bacteria in the air of some of its sewers.

The weather conditions of 1878 have been more favourable than last year for the development, multiplication, escape, and subsequent atmospheric diffusion of these sewer organisms, and these conditions have given rise to a greater prevalence of the disease and a higher fatality.

In a previous part of my paper it was shown that the above organisms are present in great numbers in the bowel-discharges and vomited matters of patients affected with the complaint under consideration. I, therefore, consider that (*a*) diarrhœa, as it affects both adults and infants during the summer months, owes its origin in the great majority of instances to the introduction of minute living organisms (bacteria) into the system by means of air or in food; and (*b*) the disease depends upon putrefactive changes in the

bowel contents, which changes are correlative to the development and multiplication of these microscopical organisms.

Let us now look at a few facts relative to the past and present of other English towns, and ascertain to what degree their experiences lend corroboration to this view of the causation of the disease.

Some twenty-five years ago very few towns possessed a complete system of drainage, and the means provided for the removal of filth were very inefficient. The inhabitants of most provincial towns were at that time surrounded by heaps of filth and other refuse, which being allowed to collect in large quantities in open vaults, attracted public attention by a more or less continuous exhalation of offensive stenches arising from putrefaction.

The close connection between a filth-contaminated atmosphere and diarrhœa prevalence was, under these conditions, fully recognised, for it almost invariably happened that, when high death-rates from the disease were experienced, there also were to be observed proportionate degrees of filth collected around the dwellings of those who had been affected.

So frequently were both conditions found to co-exist, that Mr. Simon, in his introductory report to Dr. Greenhow's paper, gave special prominence to his conviction with respect to the cause of the disease. While referring to the very unequal prevalence of diarrhœa met with in different towns, Mr. Simon says, "And any such distribution of diarrhœal disease as has just been noticed warrants a presumption—indeed, so far as I know, a practical certainty—that, in the districts which suffer the high diarrhœal death-rates, the population either breathes or drinks a large amount of putrefying animal refuse."

All the towns formerly conspicuous for high diarrhœal death-rates have now adopted and carried out various schemes to secure the more rapid removal of their filth, and so prevent its undue accumulation in their midst. The construction of general systems of sewerage supplied the means whereby these towns were enabled in great measure to accomplish this, for, in the majority of them, the greatest bulk of their excretal matter is immediately discharged through multitudinous openings into the sewers beneath.

On looking over the diarrhœal death-rates for last summer quarter of seventy English towns, as given in the quarterly report of the Registrar-General, it will be observed:

1. That all those towns, however varied their industries, remarkable for *low* death-rates, have excellent natural faci-

lities for outfall. If the advantages of site which these towns possess have been at all utilised in the construction of their sewers (and there is every reason to believe that such has been the case), then the gradients of the latter are sufficient to bestow such a velocity on the sewage flow as either insures the rapid conveyance and complete removal from beneath their inhabited areas of all the excretal matters daily received by them, or, at most, permits of the least stagnation and retention of such matters during their passage through these channels. The towns occupying such fortunate sites are, Plymouth, Bristol, Oldham, Chatham, Hastings, Southampton, Chester, Halifax, and Merthyr Tydfil.

2. That all those towns, however varied their industries, remarkable for *high* death-rates, occupy sites having a common character, viz., each area is either uniformly or in greater part flat. The facilities for outfall which their position and surroundings command, are scanty in amount and insufficient to afford such gradients to the sewers as render them self cleansing.

Some of the towns occupying such unfortunate sites are, Ipswich, Yarmouth, Leeds, Liverpool, Cambridge, Preston, Wigan, St. Helens, Leicester, and Hull.

After careful inquiry, I find that considerable excretal accumulation, resulting from insufficient fall, does actually take place in some of the sewers beneath the sites of all these towns.

I have brought under your notice the fact that the subsoil of towns during the second quarter of the year is subject to a rise in temperature, and that under ordinary seasonal changes the soil at sewer depths reaches putrefactive temperatures towards the end of June or beginning of July. During the active putrefaction of the filth stored up in these underground cesspools, the organisms giving rise to the process are thrown off in enormous numbers from the putrefying mass into the sewer air. The medium at present used to confine sewer air, viz., water, fails to arrest the outward passage of these minute forms. The bacteria in the sewer air easily pass through the water in ordinary traps and are lifted into the air by evaporation, and if the weather conditions favour this latter process, a free communication is soon established between the sewer and the atmosphere around our dwellings. Contamination of air by putrefying animal refuse is thus brought about with as great certainty, though in a less degree, as if the excretal matters were stored up on the surface and in close proximity to human habitations.

This view of the causation of diarrhœa in our large towns is, in my opinion, the only one that affords a satisfactory solution to the wide variations of mortality observed amongst their populations. It is, moreover, the one most in harmony with the convictions of Mr. Simon, a man whose great mind and most able writings place him as the first sanitarian of the world.

INFANTILE DIARRHŒA.

By GEORGE TURNER,
Medical Officer of Health to the Borough of Portsmouth.

(*Read: April 2nd*, 1879.)

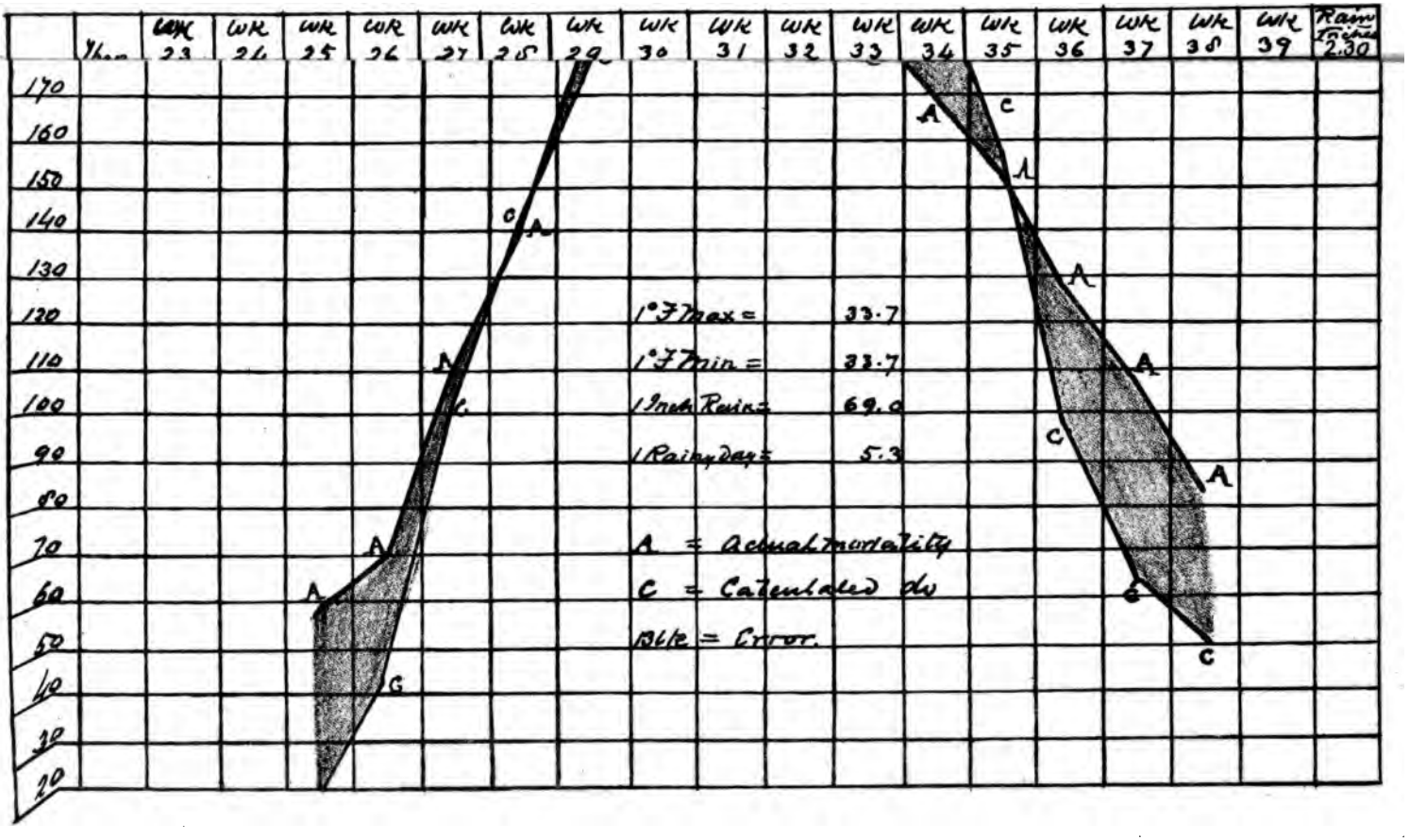
Heat has for years past been looked upon as an important factor in the production of infantile diarrhœa—by some gentlemen as the only one. This extreme view seems to have induced a certain amount of reaction. The opinions expressed by Dr. Elgar Buck, in the *Sanitary Record*, December 20th, 1878, and by Dr. Alexander McCook Weir, in the same journal, March 28th, 1879, show that there is at present a tendency to rather undervalue the effects of heat. The data, however, used by these gentlemen are totally insufficient for the purpose.*

I have recently endeavoured to put this side of the question on a firmer base, it being, I am persuaded, a point of the greatest importance in clearing up other parts of this complicated question.

I have commenced by an examination of the effect of temperature upon infantile diarrhœal mortality in London during the twelve years, 1857-68. I chose the years mentioned as I possessed Dr. Ballard's papers upon the relations of sickness and meteorology, published in the Report of the Medical Officer to the Privy Council, 1868, and I was thus saved a certain amount of labour. I have employed the annual summary of the twelve years as a starting point, and since the very great majority of the deaths from diarrhœa in the third quarter are those of infants under one year of age, I have expressed the curve in deaths per 1,000 of the births.

These averages are diagramatically represented on the accompanying table. The number of deaths actually registered are indicated by the letter A; the number arrived at by calculation, by the letter C; the error is shown by the black space between the lines. You will observe there is a deficiency which was maintained purposely, as the deaths

* The reasons were stated in the paper, but are too long for publication here.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Rain to end 2.30
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
1°F max = 33.7
1°F min = 33.7
1 Inch Rain = 69.0
1 Rainy day = 5.3
A = Actual mortality
C = Calculated dy
Blk = Error.

actually recorded [A] include those of adults as well as of infants under one year of age.

The figures of the summary are as follows:

No. of week.	Max. Fr.	Min. Fr.	Days Rain.	Ins. of Rain.	Infant Mortality from Diarrhœa.
23	70.3	50.0	2.6	0.6	21.0
24	71.0	49.0	2.1	0.3	28.6
25	74.0	50.7	2.6	0.46	57.5
26	73.5	51.7	2.6	0.4	69.5
27	73.0	51.0	2.4	0.37	108.0
28	75.3	52.3	1.4	0.2	140.0
29	74.3	54.3	2.5	0.6	183.3
30	74.0	52.5	2.0	0.65	240.3
31	73.5	51.0	2.3	0.4	229.1
32	74.5	53.3	3.4	0.7	190.2
33	73.5	53.5	2.8	0.4	183.3
34	71.7	52.3	2.8	0.75	166.1
35	71.0	51.3	2.5	0.4	152.5
36	71.3	52.3	2.8	0.45	128.0
37	66.0	49.3	2.7	0.5	100.9
38	65.0	48.7	2.7	0.4	82.4
39	65.0	48.0	3.0	0.6	66.5

Before describing the construction of the curve, I must ask you to consider the diagram headed "Theory", it is an explanation of my views as to the way in which temperature influences the death-rate with regard to time. You are asked to suppose a meteorological state of affairs for three consecutive weeks, such as to occasion for each week it continues 35-36 deaths.

From an examination of the death returns and the meteorological charts of Portsmouth, London, Yarmouth, and Leicester, I believe the greatest effect of temperature, as shown by the number of deaths, does not take place until three weeks after the occurrence of the temperature; this is plainly shown in many of the diagrams of London temperature published by Dr. Ballard, when compared with the deaths,* and on a small scale it is observable in Mr. Power's Report on the Diarrhœa in Winchester in 1876, where the following figures are given.

Progress of diarrhœa in Winchester, 1876:

No. of week.	Week ending.	No. of attacks afterwards fatal.	Deaths per week.
27th	July 8th	0	0
28th	15th	3	0
29th	22nd	2	1

* Deaths are not given in Dr. Ballard's papers, but they may be obtained from the Report of the Registrar-General.

No. of week.	Week ending.	No. of attacks afterwards fatal.	Deaths per week.
30th	July 29th	6*	1
31st	Aug. 5th	5	3
32nd	12th	5	10*
33rd	10th	5	5
34th	26th	0	4
35th	Sept. 2nd	0	3
36th	9th	0	1
37th	16th	0	0
38th	23rd	0	0
39th	30th	1	0
40th	Oct. 7th	0	1

A further consideration of the figures of the summary of the twelve years has induced me to believe that this does not express the whole truth, and that a portion of the children die during the week of high temperature, a few more the week after, the majority, as before said, during the third week; and that there is a residuum which dies on the fourth week, and even still later. It should be remembered I here refer to deaths *registered* as due to diarrhœa; the relation between these tardy deaths and infantile diarrhœa may, perhaps, be a proper subject for consideration.

Bearing in mind, then, the conditions named in the heading of the diagram, I have arrived at the conclusion that of the deaths due to each week's influence,

$\frac{1}{7}$	will die	during	the 1st	week	= 5
$\frac{2}{7}$	,,	,,	2nd	,,	= 10
$\frac{3}{7}$	,,	,,	3rd	,,	= 15
$\frac{1}{9}$	,,	,,	4th	,,	= 4
$\frac{1}{36}$	,,	,,	5th	,,	= 1

And so on, dividing by four.

The total mortality registered in each of the three weeks, therefore, will be:

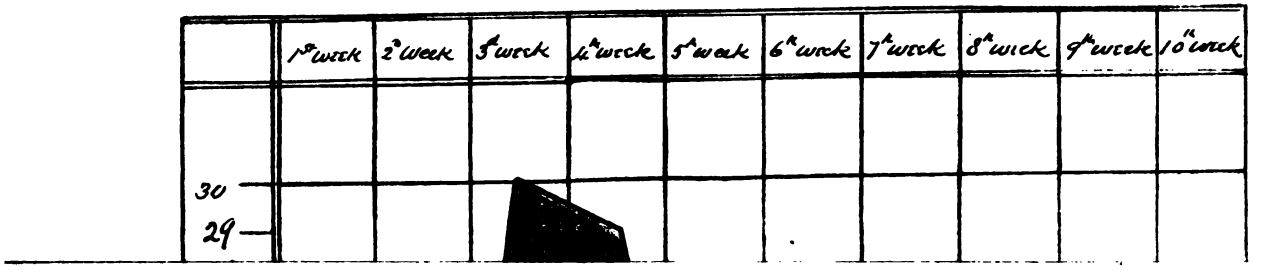
1st week.	2nd week.	3rd week.	4th week.	5th week.	6th week.	7th week.
5	10	15	4	1	etc.	
	5	10	15	4	1	etc.
		5	10	15	4	1
5	15	30	29	20	5	1 = 105

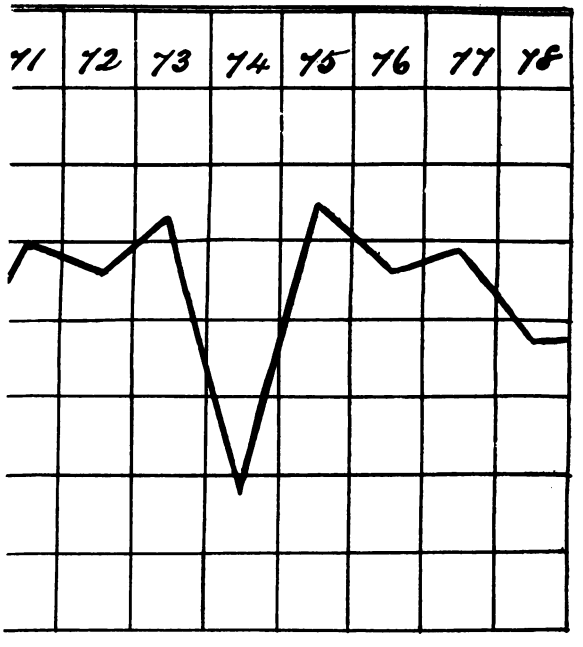
In the accompanying diagram the white space represents the deaths due to the first week, the shaded space those due to the second week, and the darkest portion those due to the third week.

* The date of attack could not in all cases be ascertained.

Theory

Atmospheric conditions for three weeks such as to produce 35 – 36 Deaths The following weeks bring neutral.

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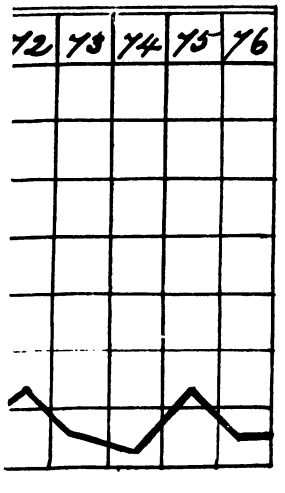
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This opinion, as to the incidence of the greatest number of deaths upon the third week after the temperature causing them, I first put forward in my Report to the Urban Sanitary Authority Portsmouth six years ago. I am tolerably sure it is correct as far as regards England, but am not so confident respecting the course of events in America. The proportions here laid down were arrived at during the construction of the curves in the first diagram, which has reference exclusively to London. As regards the formation of these curves I was guided by the following rules:

1. Other necessary causes being present in a town, diarrhœa is produced whenever the weekly mean of the daily minimum temperature is above 50 deg. Fahr., but to produce any very appreciable effect it must continue for three consecutive weeks; many instances proving the statement are in my possession, and if a curve were constructed for two consecutive weeks on the same principles as those employed in forming the accompanying theoretical one, instead of 105 deaths you would only have 30. In London, 1 deg. Fahr. above 50 deg. F. is equivalent to 33.7 deaths per 1,000 births.

2. When the minimum temperature has reached 50 deg. the maximum temperature begins to bear its part, and each degree of weekly maximum temperature above 70 deg. Fahr. produces an equal effect, that is to say, it produces in London deaths equal to 33.7 per 1,000 births. I think a glance at the diagram of mortality recorded in Winchester during the years 1867-78 will explain my meaning, you will there see that during 1867, 69, 70, 71, 72, 73, 74, 75, 77, and 78 the mortality from this particular cause does not average 1 per cent. of the births, but that it is trebled in 1868 and 1876. Concerning 1868, I only know generally that it was an excessively hot and dry summer, which was accompanied by a general increase in the diarrhœa rates all over the country; but for 1876 we have the observations taken by Dr. Earle of Winchester. Compare them with those taken by the same gentleman for the years 1873, 74, 75, 77, and 78, and observe the continued rise of the minimum temperature above 50 deg. in the year 1876. That the minimum temperature is the most important is shown by the accompanying temperature chart for Great Yarmouth—a town unfortunately remarkable for this infantile disorder, but where the weekly mean of the maximum temperature rarely reaches 70 deg. Fahr. The observations from which these tables were constructed were taken by Mr. Weston, of the Sailor's Home, Yarmouth, to whose kindness I am indebted for them.

3. Rain diminishes the death-rate, not only by cooling the air—if this were its sole action it might be left out of con-

sideration, the thermometer might be allowed to record its influence—but it is potent for good in other ways, chiefly, I believe, by purifying the atmosphere, probably in a mechanical manner.

4. It is important to note, not only the quantity of rainfall, but also the number of days on which it falls. In London I found that one inch of rain diminished the death-rate by 62.0 per thousand of the births, but every day on which it fell increased this quantity by 5.3, thus one inch falling in one day diminished the death-rate by 74.3, but if it fell in seven days this number was increased to 106.

In making the curve (London summary of 12 years) I did not actually use the constant 33.7 for the temperature, dividing the deaths up with sevenths, ninths, thirty-sixths, etc. I used a constant 14.5 for the deaths of the third week after the temperature, taking $\frac{1}{3}$ as the deaths of the first week, $\frac{2}{3}$ as the deaths for the second week, and $\frac{1}{4}$ as the deaths for the fourth, continually dividing by four.

$$\frac{14.5}{3} + \frac{14.5 \times 2}{3} + 14.5 \quad \frac{14.8}{4} = 33.7$$

And so on for the other values.*

This was done because I had already constructed the tables before I clearly saw their meaning. The result is the same, and, besides wasting my time, it would have rendered the figures I now place before you much more complicated.

Summary of twelve years figures for Curve.

Week, 25th = 4.7 deg. Fahr. by 14.5 = 68.1.

0.45 Inches Rain by 20.6	= 13.32	68.1		
2.6 Days Rain by 2.257 =	5.86	19.1		
	19.18	49.0		16.3 = 25th week.
		22.7 = $\frac{2}{3}$		
		16.3 = $\frac{1}{3}$		

Week, 26th = 5.2 deg. Fahr. by 14.5 = 75.4.

0.4 Inches Rain by 29.6	= 11.84	75 4		
2.6 Days Rain by 2.257 =	5.86	17.7	22.7	$\frac{2}{3}$ of 25th Week
	17.7	57.7	19.2	$\frac{1}{3}$ of 26th Week
		38.5 = $\frac{2}{3}$		
		19.2 = $\frac{1}{3}$	42.9 = 26th Week Mort.	

Week, 27th = 4.0 deg. by 14.5 = 58.0.

0.37 Inches Rain by 29.6	= 10.95	58.0	49.0	26th Week.
2.4 Days Rain by 2.257 =	5.41	16.3	38.5	$\frac{2}{3}$ of 26th Week
	16.36	41.7	13.9	$\frac{1}{3}$ of 26th Week
		27.8 = $\frac{2}{3}$	101.4 = 27th Week Mort.	
		13.9 = $\frac{1}{3}$		

* The constant for an inch of rain was 29.6, that for a day's rain 2.257.

Week, 28th = 7.6 deg. by 14.5 = 110.0.

0.2 Inches Rain by 29.6	= 5.92	110.0	25.3	¼ Mrt. in Wk. pcdg.
1.4 Days Rain by 2.257	= 3.15	9.0	57.7	26th Week
			27.8	⅔ of 27th Week
	9.07	101.0	33.6	⅓ of 28th Week
		66.4 = ⅔		
		33.6 = ⅓	144.4	= 28th Week Mort.

Week, 29th = 8.6 deg. by 14.5 = 124.7.

0.6 Inches Rain by 29.6	= 17.76	124.7		
2.5 Days Rain by 2.257	= 5.64	23.4	36.1	Figures as in former weeks
			41.7	
	23.4	101.3	66.4	
		67.6 = ⅔	33.7	
		33.7 = ⅓		
			177.9	= 29th Week Mort.

Week, 30th = 6.5 deg. by 14.5 = 94.2.

0.65 Inches Rain by 29.6	= 19.24	94.2	44.4	
2.0 Days Rain by 2.257	= 6.15	23.7	101.0	
			67.6	
	23.75	70.5	23.5	
		47.0 = ⅔		
		23.5 = ⅓	236.5	= 30th Week Mort.

Week, 31st = 4.5 deg. by 14.5 = 65.2.

0.4 Inches Rain by 29.6	= 11.84	65.2		
2.3 Days Rain by 2.257	= 5.20	17.0	59.1	
			101.3	
	17.04	48.2	47.0	
		32.1 = ⅔	16.1	
		16.1 = ⅓		
			223.5	= 31st Week Mort.

Week, 32nd = 7.8 deg. by 14.5 = 113.0.

0.7 Inches Rain by 29.6	= 20.72	113.0		
3.4 Days Rain by 2.257	= 7.67	28.3	55.8	
			70.5	
	18.39	84.7	32.1	
		56 5 = ⅔	28.2	
		28.2 = ⅓		
			186.6	= 32nd Week Mort.

Week, 33rd = 7.0 deg. by 14.5 = 101.5.

0 4 Inches Rain by 29.6	= 11.84	101.5		
2.8 Days Rain by 2.257	= 6.32	18.2	46.6	
			48.2	
	18.16	83.3	56.5	
		55.5 = ⅔	27.8	
		27.8 = ⅓		
			179.1	= 33rd Week Mort.

Week, 34th = 4.0 deg. by 14.5 = 58.0.

0.75 Inches Rain by 29.6	= 22.20	58.0		
2.5 Days Rain by 2.257	= 5.65	27.9	44.7	
			84.7	
	27.85	30.1	55.5	
		20.1 = ⅔	10.0	
		10.0 = ⅓		
			194.9	= 34th Week Mort.

Week, 35th = 23 deg. by 14.5 = 33.4.				
0.4 Inches Rain by 29.6	= 11.84	33.4		
2.5 Days Rain by 2.257	= 5.65	17.5		48.7
	17.49	15.9		83.3
				20.1
			10.6 = ⅔	5.3
			5.3 = ⅓	
				157.4 = 35th Week Mort.
Week, 36th = 3.6 deg. by 14.5 = 52.2.				
0.45 Inches Rain by 22.6	= 13.32	52.2		
2.8 Days Rain by 2.257	= 6.32	19.6		39.3
	19.64	32.6		30.1
				10.6
			21.8 = ⅔	10.8
			10.8 = ⅓	
				90.8 = 36th Week Mort.
				22.7
				15.9
				21.8
				60.4 = 37th Week Mort.
				15.1
				32.6
				47.7 = 38th Week Mort.

Of course these figures do not pretend to mathematical accuracy, the values were found experimentally. They may not be applicable to London for any other period of twelve years; they certainly are not for most other towns, because the numbers used are compounded of three or four variables and at least one constant.

For instance, no one doubts that artificial feeding at least assists in producing the disease; no one disputes the influence of dirty habitations, overcrowding, bad drainage, etc., or of a polluted water-supply, the degree only in which these circumstances influence the mortality is the subject of dispute.

Confining our attention for the present to the effects of temperature alone, its influence evidently must be the same everywhere. One degree of temperature alone can perform no more in one part of England than in another; but I can conceive the effect on the inhabitants, produced by its action on surrounding conditions, may be various.

Before proceeding farther, I must ask you not to look at the following formulæ from a strictly mathematical point of view. Let the effect of temperature be represented by x, the amount of artificial feeding by a, the effect of the sanitary condition of the place by b, the effect of the water-supply by c. Then in London the value, 33.7 is composed of x (a, b, c, etc.), now x must be a constant, but a, b, c, etc., may

vary indefinitely, and may be represented by a', b', c', etc., or a'', b'', c'', etc.; then instead of 33.7 we may have a value represented by x (a', b', c', etc.) or x (a'', b'', c'', etc.)

To illustrate my meaning more fully confine your attention to the first variable, *i.e.*, the effects of artificial feeding represented by a, either remove the other variables entirely from consideration or let them be represented by unity, which in the case of a multiplier amounts to the same thing. Conceive three towns in which the populations of infants artificially fed are respectively 25, 50, and 75 per cent., or as 1, 2, and 3, then the variable for the respective towns, which must be applied to the temperature in calculating its influence, must be as 1, 2, and 3.*

If this method of inquiry were applied to different towns the actual value of x might perhaps be obtained, the other components being massed together and regarded as a sort of personal equation representing the outcome of the other influences existing in the town, and a much better idea of the value of sanitary improvements would be thus obtained.

I have been unable to work out the death-rate week by week for the different years for various reasons, but I have made an attempt, a rough one it is true, to reproduce the curve of the years.

If the following facts are remembered a constant relation can be observed.—1. Three weeks of minimum temperature over 50 degs. Fahr. are required to produce an effect.—2. After three weeks' minimum temperature above 50 degs. Fahr., each degree at average maximum temperature above

* I have been careful to state that the symbols employed above must not be employed in a strictly mathematical sense. You see that I have employed a sign indicating multiplication; I ought to state that two gentlemen, whose experience lends weight to their opinions, are inclined to think the sign of multiplication should give place to that of addition—one of them, a celebrated statist, has kindly promised to consider the question as soon as this is printed, and will endeavour to find a formula which shall be mathematically correct. As my own knowledge is, unfortunately, too small to enable me to decide such a question, I shall content myself with stating the reasons which induced me provisionally to employ the sign of multiplication. Any series of figures connected by plus signs can be equally well represented by corresponding figures greater than unity, united by the sign of multiplication, and should minus signs have been employed in any case, by figures less than unity. There are places where in summer the heat reaches a point above that necessary for the production of diarrhœa, but which do not suffer from this disease, because the other factors are absent, and occasionally, as I believe, because the principal one, infant feeding, is absent. Now, to produce such a result without the sign of multiplication, the plus sign must be changed to minus; when the sign of multiplication is used no such necessity exists; the total absence of this factor, without which heat, etc., is impotent, has only to be represented by zero to produce the result.

70 degs. must be conceded.—3. Breaks in the continuity of the average minimum temperature above 60 degs. Fahr., interfere with the result, roughly estimated at about $\frac{1}{6}$, the amount obviously depends upon the fall of the temperature and the length of time it continues.—4. Rainfall as measured by its effect is equivalent to 2 degs. Fahr., for every inch of fall, with a minus value.—5. Each rainy day equals $\frac{1}{12}$ inch rainfall or $\frac{1}{6}$ Fahr., with a minus value.—6. The average diarrhœa mortality for the 1st, 2nd, and 4th quarters is 2.7, a number consequently independent of temperature, and which must be deducted from the average before making the calculation and must subsequently be added.

I have added together the number of degrees of temperature recorded above 50 degs. Fahr., and added them to those above 70 degs. Fahr., and have considered them as a superficies, as a space plotted out in a diagram, then taking the summary as the standard of comparison, the death-rates equal to the square roots; but the rainfall has to be deducted, this I have done by multiplying every inch of rainfall by 2, and dividing the number of rainy days by six, and adding them together; it is apparent at the first glance that if this line of reasoning were rigidly carried out the one should be deducted from the other. I have been unable to do this, the best result I have been able to arrive at is by the following process:—

$\sqrt{143 \div 65.8} \times 18.1^{*}$	= 26.6
$\sqrt{23.4 \div 16.6}$	= 1.3
Average	25.3
Average of the other quarters	2.7
Calculated mortality	28.0
Actual ditto	26.0

Whenever there was a break in the continuity of the minimum temperature above 50 degs. Fahr. I have deducted $\frac{1}{6}$; and in this way I have constructed the opposite table, in which the actual mortality is indicated by A, the calculated mortality by C, and the error by the space shaded black.

* The yearly summary gives 19.8, from which the average of the other three quarters, 2.7, being deducted, 17.1 remain; the number 18.1 has been employed because, by the formula given above, a year exactly resembling the average would give 16.1 instead of 17.1.

The actual figures from which the table has been made are these :—

1857.

$\sqrt{143.0 \div 65.8} \times 18.1$	=	26.6
$\sqrt{23.4 \div 16.6}$	=	1.3
		25.3
Average, 1, 2, and 4 qrs.		2.7
		28.0
Calculated		28.0
Actual		26.0

1858.

$\sqrt{130.9 \div 65.8} \times 18.1$	=	25.6
$\sqrt{19.0 \div 16.6}$	=	1.0
		24.6
3 Breaks		12.3
		12.3
Average, 1, 2, and 4 qrs.		2.7
		15.0
Calculated		15.0
Actual		15.5

1859.

$\sqrt{160.8 \div 65.8} \times 18.1$	=	28.0
$\sqrt{14.0 \div 16.6}$	=	0.9
		27.1
Average, 1, 2, and 4 qrs.		2.7
		29.8
Calculated		29.8
Actual		29.6

1860.

$\sqrt{5.5 \div 65.8} \times 18.1$	=	5.2
$\sqrt{7.8 \div 16.6}$	=	.7
		4.5
Average, 1, 2, and 4 qrs.		2.7
		7.2
Calculated		7.2
Actual		8.0

1861.

$\sqrt{99.9 \div 65.8} \times 1.81$	=	22.3
$\sqrt{17.0 \div 16.6}$	=	1.0
		21.3
Average, 1, 2, and 4 qrs.		2.7
		24.0
Calculated		24.0
Actual		19.2

1862.

$\sqrt{22.6 \div 65.8} \times 18.1$	=	10.6
$\sqrt{12.2 \div 16.6}$	=	0.8
		9.8
1 Break		1.6
		8 2
Average, 1, 2, and 4 qrs.		2.7
		10.9
Calculated		10.9
Actual		9.8

1863.

$\sqrt{31.8 \div 65.8} \times 18.1$	=	12.6
$\sqrt{4.4 \div 16.6}$	=	0.5
		12.1
Average, 1, 2, and 4 qrs.		2.7
		14.8
Calculated		14.8
Actual		17.2

1864.

$\sqrt{55.6 \div 65.8} \times 18.1$	=	16.6
$\sqrt{6.0 \div 16.6}$	=	0.6
		16.0
Break		2.7
		13.3
Average, 1, 2, and 4 qrs.		2.7
		16.0
Calculated		16.0
Actual		19.8

1865.

$\sqrt{116.6 \div 65.8} \times 18.1$	=	23.5
$\sqrt{20.6 \div 16.6}$	=	1.1
		22.4
1 Break		3.8
		18.6
Average, 1, 2, and 4 qrs.		2.7
		21.3
Calculated		21.3
Actual		20.5

1866.

$\sqrt{76.5 \div 65.8} \times 18.1$	=	19.5
$\sqrt{28.0 \div 16.6}$	=	1.3
		18.2
Average, 1, 2, and 4 qrs.		2.7
		20.9
Calculated		20.9
Actual		21.8

1867.		1868.	
$\sqrt{52.9 \div 65.8} \times 18.1$ =	16.2	$\sqrt{177.3 \div 65.8} \times 18.1$ =	29.7
$\sqrt{6.8 \div 16.6}$ =	0.6	$\sqrt{11.8 \div 16.6}$ =	0.8
	15.6		28.9
Average, 1, 2, and 4 qrs.	2.7	Break	4.8
	18.3		23.1
Calculated		Average, 1, 2, and 4 qrs.	2.7
Actual	19.5		
		Calculated	25.8
		Actual	27.6

These results are sufficiently near the truth to demonstrate the controlling effect of temperature and to justify the assertion I made in my report for the year 1877, that—"Given a certain percentage of infants in a town, who receive other nourishment than breast milk, the general state of the town being the same, the mortality from diarrhœa will be entirely ruled by meteorological conditions"; this assertion you will find contested both by Dr. Elgar Buck and Dr. McCook Weir, in the *Sanitary Record*.

I believe I shall shortly be able to find a formula for calculating the yearly mortality which shall be free from the objections I have pointed out in the one now employed: as, however, the principle would be the same, and my time is short, I have contented myself with this approximation.

I find that the question of age in relation to diarrhœa requires to be re-considered. In 1874 I found that

10.3	per cent. of the cases were aged	1	month and under
11.3	,, ,,	2	,,
14.2	,, ,,	3	,,
5.1	,, ,,	4	,,
6.1	,, ,,	5	,,
3.0	,, ,,	6	,,
3.0	,, ,,	7	,,
6.0	,, ,,	8	,,
2.0	,, ,,	9	,,
4.1	,, ,,	10	,,
8.2	,, ,,	11	,,
25.7	,, ,,	12-24	,,

In Leicester, Dr. Buck and Mr. Franklin found—

10.8	per cent. aged	1	month and under
23.0	,,	1-3	,,
26.8	,,	3-6	,,
24.0	,,	6-12	,,
13.6	,,	1-8	years

London.

Mortality from Diarrhœa in the 3d Qrs

Deaths	1857	1858	1859	1860	1861	1862	1863	1864	1865	1866	1867	1868
34												
33												
17												
0												

While Dr. Sloane, in the same town, found the following per centage:—

	Under 1 year.	1–2 years.	2 years and upwards.
1875	78	13.8	8.2
1874	85	11.4	3.6
1873	80	14.0	6.0
1872	72	17.0	11 0
1871	80	11.8	8.2
Average	69	13.6	7.4

The ages being differently arranged are not strictly comparable; it is, however, evident that the numbers dying under six months in Leicester are greater than the numbers found in Portsmouth. Dr. Johnston, in his report for 1877, does not divide the ages into months, but he found that 84 per cent. of the deaths occurred under one year of age.

The figures just quoted, it may be incidentally remarked, reduce the effect which teething has on the disease to a very insignificant position in the causation of this disease. They were accepted as conclusive evidence that the disease was essentially an infantile disorder, until Dr. Johnston, in the course of his extended enquiries at Leicester, caused the ages of persons applying for medicine for the relief of diarrhœa to be recorded; he then found that of 3318 cases 1590 were children under five years of age, and 1728 were adults and children above five years of age. On discovering this he wrote: "These figures clearly indicate that the disease is more prevalent amongst adults than amongst children." It must not be overlooked that the gratuitous distribution of medicine is not a good test as to the prevalence of any particular disorder: those who have had any experience of the working of a charitable institution must be aware there are numbers of persons who will take physic whenever they have an opportunity, and they will appreciate the extent to which this disposition is likely to vitiate the returns. But even if we accept Dr. Johnston's figures as correctly representing the prevalence of the disease at the ages specified, they contain the refutation of his inference; the numbers 1590 and 1728 do not by any means represent the proportion of persons living under five years and over five years of age in Leicester; but making a calculation based on the whole population of Leicester, a proceeding manifestly in favour of his view, they would represent respectively 9.8 per cent of the persons under five years of age, and 1·7 per cent of those over five years old.

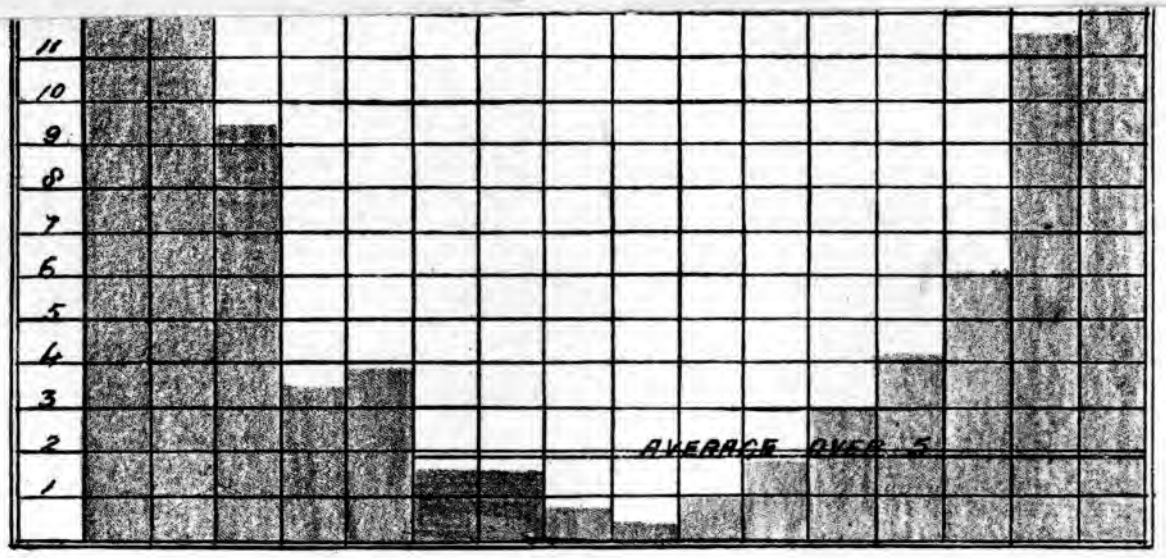
I thought it worth while, however, to obtain more informa-

tion on this point: I accordingly searched the books of the Poor Law Medical Officers of Portsmonth, Portsea, Southsea, and Landport, and extracted all the cases of diarrhœa which were entered during the third quarters of the last six years. I arranged them in sixteen groups of ages; I next ascertained the number of paupers in the Borough on the first day of July, during the last ten years, and took the average. I calculated the numbers living at the specified ages, supposing the pauper population resembled the general population as to the age distribution, and then obtained the percentage of cases to persons living. The numbers thus obtained are probably not strictly accurate, but they are certainly nearer the truth than those previously quoted. I have tabulated the results; the percentage of cases to children under five years of age was 14.5, and the percentage of cases over five years of age to the populations was 0.8; the figures for the last decade are not reliable, the numbers on which they were based being too small.

The table clearly shows that the disease is most prevalent at early ages, and although we may reasonably infer it again appears to prevail in extreme old age, when the digestive system is weaker, we must not overlook the existence of diarrhæa in the old as a symptom of many other disorders.

I have now to consider the alleged connection between defective drainage and diarrhœa. Dr. Johnston has found that the majority of deaths in Leicester occurred in drained houses, and expresses the opinion that conditions favourable to typhoid fever also produce this disease; there is, I believe, one other medical officer of health who has similar views. I did not know the relative number of drained and undrained houses in Leicester, consequently I cannot utilise Dr. Johnston's observations. In 1875 I examined into 122 fatal cases of diarrhœa, 46 occurred in drained houses, *i.e.*, 37.7 per cent. Now at that time the number of inhabited houses in Portsmouth was 20,219, and of these 8,176 were drained, or 40.7 per cent., so that the drainage in this instance at least seems to have affected the question very little.

I have since then examined the point more closely, and I have marked off on a map all the cases of diarrhœa which have occurred in the third quarters of the last six years in Portsmouth; I have not published the map, as a verbal description will be readily understood. It is immediately apparent that the greatest number of fatal cases occur in the towns of Portsmouth and Portsea, and in that portion of the Borough that lies to the north of Somer's Road.


11
10
9
8
7
6
5
4
3
2
1
AVERAGE OVER 5

In Leicester, the mortality was represented as occurring chiefly in the low-lying parts of the town; I have consequently taken the surface levels on sections traversing the localities in which the greatest number of deaths occurred, and also those in which there were the fewest, and I have represented them in the accompanying diagram; the straight line is the ordinary high-water mark, and the round marks represent the sewers; the result is, that in Portsmouth, unlike Leicester, the greatest mortality occurs in the highest parts of the town, where the sewers are never blocked, as is the case at every tide with all those which are situated below the high-water mark.

It is also true that the town south of Somer's Road is not so densely populated as the northern portion, and it might fairly be objected that the number of deaths, as indicated by the figures in the map, is not a correct measure of the mortality. To remove this objection, I have counted the number of houses on each side of the section to the distance of 500 feet; I have multiplied the number so found by the average number of persons found in each house at the last census, and in this manner have endeavoured to estimate the populations at different points: the results are shown on the diagram by the shaded surface, which indicates that the mortality of Portsmouth, Portsea, and the north of Somer's Road, is double that of the rest of the town. Our sewers, as you are aware, are ventilated at frequent intervals into the roads; yet in localities where during every high tide the sewer is filled with stagnant sewage, which necessarily gives off more or less gas, the inhabitants suffer less from this disorder. In Stamshaw, where there is no sewer, the disease is prevalent and very fatal; nor can I find any constant relation between typhoid fever and diarrhœa.

I do not wish it supposed that these facts lead me to deny that a defective sewer has any influence, I only contend that it is not *the influence* which is to be sought for. Let Dr. Johnston's improvements be carried out, and I have no doubt they will exert a beneficial effect on the mortality from diarrhœa in common with that produced from all other disorders, but I do not think they will suffice to take Leicester out of the category of towns with an abnormally high diarrhœal death-rate: I should certainly expect the most marked effect would be seen in the deaths from typhoid fever.

It might fairly be asked to what do I attribute the

marked differences seen in the mortality from diarrhœa, in certain portions both of Portsmouth and Leicester?

He says, "This table goes to show how exactly deaths from diarrhœa compare with the density of population and unhealthy areas. Take for example the north district, and you will find that with a population 7000 fewer than that of the west district you have 117 deaths against 84; but then comes the secret that the north has 31 more souls than the west upon every acre of its area, and the result is that the north scores a death-rate of 2.14 per 1000 from diarrhœa, whereas the west district registers only 1.2 per 1000. I need not dictate further on the co-relation of density of population per acre, and the mortality rate from diarrhœa; for if the table appended hereto be carefully studied it will be seen how almost exactly to a decimal this fact is curved out."

DIARRHŒA IN LEEDS.

Dr. Goldie, of Leeds, in his Report for 1876, published the following Table:—

Registration Sub-Districts of Leeds Borough.	Estimated population in the middle of the year 1876.	Number of Persons living per acre.	Total deaths from Diarrhœa during 1876.	Death rate per 1,000 per annum living.	Births registered during four quarters of the year.				Total Deaths.
					1st.	2nd.	3rd.	4th.	
Leeds Township-									
South-East -	29,071	34.0	51	1.07	358	352	304	321	1335
North -	54,582	85.3	117	2.14	657	586	565	583	2391
West -	67,616	54.3	84	1.2	682	652	658	620	2612
Hunslet - -	45,250	39 12	83	1.9	485	527	474	485	1971
Holbeck - -	20,847	7.8	39	1.8	220	242	258	242	962
Wortley - -	37,414	9.1	52	1 4	414	427	330	378	1549
Bramley - -	10 568	4 2	20	1.8	118	98	85	109	410
Kirkstall - -	16,888	5 3	26	1.6	144	165	140	166	615
Chapel Town -	8,962	11.6	5	0.6	66	71	75	60	272
Whitkirk - -	482				9	5	2	1	17
Total of Borough	291,580	111	478	1.35	3153	2891	2891	2965	12,134

After examining the table carefully, I think this is claiming too much; the only case in which we get an appropriate result is when we compare the north and the west district—all the others are very far out; and this result is only obtained by comparing the number of persons living on an acre. This would indicate that density of population exerted an enormous influence, especially when it is remembered that its effect upon the general mortality is only measured by the eighth root of the density. The influence

of density cannot be shown by such areas as Dr. Goldie has chosen. I hope soon to be able to publish some observations of this side of the question, but they can only be obtained by attention to the social position of the people living in the district. I believe that the rate-books will in this matter be our best available guides.

The deaths in Portsmouth are principally confined to that portion of the town inhabited by labourers and mechanics, and the deaths occur almost exclusively amongst persons in that class of life. In the localities inhabited by richer persons, the deaths occur in little islands of houses (if I may use the term) inhabited by the same class. The children of the rich suffer from this disorder, but it is not so fatal, they have earlier medical attention, and the parents are in a position to take greater care of them.

I do not for a moment dispute the importance of good sanitary conditions, whether connected with the drainage or the cleanliness of the houses and their inhabitants. I do not deny the possibility of diarrhœa from the inhalation of foul air; to do so would be to fly in the face of experiment and general experience; indeed, I drew the attention of the Portsmouth Sanitary Authority to this point in 1874; I believe not only that bad air, but air such as under the best circumstances we must expect in towns, and which we find in the country under unfavourable conditions, exerts its influence much more easily through the food.

In Leicester, the introduction of new industries, on two occasions increased the infantile diarrhœa mortality, while the partial destruction of the Coventry ribbon trade reduced it; but I wish particularly to refer to some criticisms upon my remarks upon the mortality from diarrhœa in Coventry.

In the diagram representing the diarrhœa in Coventry, you will observe an enormous decrease in the mortality after 1861, and this decrease I attributed mainly to the decline of the ribbon trade, consequent upon the treaty of 1860—(it is true that the drainage was commenced in 1857, but it took years to connect the houses, and I am informed that although the water-supply was commenced in 1846, it was not generally used until 1863); while in 1870 you will remark an increase, just when the Franco-German War gave an impetus to the Coventry ribbon trade.

Dr. Johnston doubted the possibility of the French War exerting any influence in 1870, he thought it broke out too late. Since he read his paper before the Epidemiological Society, however, I have made enquiries in the ribbon trade, and I find it is true that the greatest pressure was

put upon the Coventry trade at a time when it could not have affected the disease. The heaviest orders are given at the end of the year for the spring trade, but there is also a second period of great activity in the summer, for the autumn trade. Now, fighting between France and Germany actually began on July 19th, although war was not declared until next day, and so rapid was the progress of disorganization, that the Republic was declared in Lyons in September. Besides, the very probability of war seems to have affected Coventry, for we find trade was remarkably brisk even before it was declared. Dr. Mark Fenton, in a letter to me says: "As regards Coventry the evidence is all in your favour, but I think we require some further investigation into the subject to account for the disease in the whole kingdom."

I would ask any medical man practising among the poorer classes, if amongst those below the position of mechanics, who are prevented by poverty from using artificial food, he does not find an immunity from summer diarrhœa amongst the children, although they are otherwise unfavourably situated?

Two most important questions I must leave undiscussed; one of them is the connection supposed by some to exist between diarrhœa and an impure water-supply, for which I believe there are good grounds; although I see that one medical man has objected that children under one year of age do not drink water, overlooking the fact that it is largely employed in artificial feeding. I am sorry to see that my remarks upon the water-supply in Yarmouth (published in my yearly report for 1877), have given offence to the Authority there. I am aware that the company has been doing much to supply a better water than that obtained from the wells in the town; that the water from the "Broad" is the best available is unfortunate (although it is undoubtedly preferable to the well water), but even when judged by the analysis supplied by the company, there is still much to be desired. I do not suppose it is contaminated by sewage, nor do I suppose such a contamination is necessary for the production of this disease.

The second question is that raised by Dr. Buchanan, at the Society of Medical Officers of Health, as to the occurrence of this disease in the metropolis in the 17th century, its disappearance in the 18th, and its re-appearance in the 19th. These are both wide questions, and for the second especially I lack the necessary information.

I attribute the disease to the introduction of matters

undergoing fermentation or putrefactive changes into the susceptible intestine of a young child and a continued high temperature.

I have already on former occasions considered this question, and shall not do so now beyond noticing that Dr. Johnston in his paper on this subject, which should be studied by every medical officer, has attributed the disease to bacteria.

I have been careful not to lay the blame entirely upon the *toruloid spores* or upon the presence of *bacteria;* it is by no means clear to me that the acidity of the food is not the principal cause, and although it may be objected that without the presence of spores milk would not be acid. I

ERRATA.

Page 220, line 8, *for* "62.0 per thousand" *read* "69.0".

Page 222, line 2 from bottom *et seq.*: *for* x $(a, b, c,)$, *read* x $(a . b . c.)$, etc.

Page 224, line 1, *for* "conceded", *read* "counted".

Page 230, line 3, the paragraph "He says," etc. should follow the Table headed "Diarrhœa in Leeds—Dr. Goldie," etc.

Page 230, line 8 from bottom, *for* "appropriate", *read* "approximate".

REMARKS ON THE ORIGIN OF INFECTION.

By R. THORNE THORNE, M.B., F.R.C.P. Lond.,
Medical Inspector to the Local Government Board.

(*Read: April 8th*, 1878.)

I PURPOSE to-night bringing under your notice a few points relating to the origin of infection. The question is one which has, more or less, at all times occupied the attention of the epidemiologist, but it has, in its varied aspects, been specially forced upon him during recent years, and as some time has now elapsed since the subject was discussed by this Society, I have thought that there might be some advantage in reading a brief paper with a view of eliciting opinions on a topic, as to which many present are, by the nature of their several experiences, specially able to speak with authority.

For obvious reasons I shall make no attempt to deal with the subject exhaustively; the mere time at my disposal would alone frustrate any such attempt. But besides this, any effort to handle the question in its numerous aspects, physiological, biological, chemical and other, would be completely beyond the range I have set for myself, which is, to regard the subject from the point of view of one who, having but little acquaintance with the laboratory, has to rely for most of his data on experience gained in what I may perhaps term the field-work of epidemiology.

In the remarks which I propose to make, I desire specially to hold in view this question: Can any case of acute specific fever arise independently of an antecedent case? And by the term acute specific fever, I mean all acute febrile diseases capable of reproducing their own kind. With a view to the elucidation of this question I will, in the first instance, attempt a review of the principal grounds which might lead some observers, on the one hand, to answer it in the negative, others, on the other hand, to reply affirmatively.

I. The following arguments might be brought forward by those who, at times termed contagionists, maintain that no case of infectious disease can arise independently of a prior case of the same disease.

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1. There is the general inference to be drawn from the well-known fact that infectious diseases do largely owe their spread to communication, immediate or mediate, with prior cases of the same disease. With regard to many of these diseases we know what are at least the ordinary means by which their poisons are eliminated from the bodies of those affected by them, and these poisons we can trace, at times, through numerous and intricate channels, until at last we find that in some way or other they are received into the systems of some hitherto healthy persons, in whom forthwith the diseases they give rise to are reproduced with all their well-known signs and symptoms. This, then, being admittedly the ordinary source of infection, surely, it may be urged, there are strong *à priori* grounds for assuming it to be the invariable one; for even when direct evidence as to the transmission of the specific poison cannot be traced, there must be ample reason for assuming that in some hitherto undetected way the same process has been followed out. Indeed the alternative view is, it may be alleged, founded solely on negative evidence, and is based on nothing better than the failure of the investigator to trace out a succession of circumstances, often obscure, but which are well known to have existed in many other similar instances.

2. And this first argument is strengthened by consideration of the fact that new channels by which the poisons of these specific diseases can be conveyed from one person to another are periodically being discovered; and that consequently outbreaks, which formerly would have been brought forward as affording indubitable evidence as to spontaneous origin, on the ground that all known sources of the diseases in question could with certainty be excluded from the list of possible causes, are now known to have been due to the conveyance of the poison by means of some hitherto unknown channel. Thus since it was ascertained that enteric fever and scarlet fever could be transmitted through the agency of milk as a vehicle, numerous outbreaks of these diseases, especially of enteric fever, have been ascertained to have owed their spread to this means, and thus that which must formerly have remained unexplained on the theory of the contagionist, now tends to confirm his views. The same remark applies to the striking outbreaks of enteric fever which have owed their spread to that form of water-pollution which in intermittent water-services leads to the specific poison of the disease being drawn with other filth into the water-mains during periods of intermission,

and to its being subsequently distributed to the general body of consumers. And if milk can thus act as a vehicle for specific infection, and if water can thus in this newly-discovered method transmit the poison of enteric fever, may not other articles of food act in a similar manner; and may not water be a carrier of infection by other and, as yet, unknown means? In short, is it not likely that other channels for the communication of infection may be discovered, and that in this way all doubt as to the possibility of the spontaneous origin of the diseases under consideration may, step by step, be removed? And hence, where all known methods for the conveyance of these poisons fail to explain the existence of the diseases produced by them, is it not fair to assume that the infection has really been communicated, though by some hitherto undetected channel?

3. In cases, too, where the history of an attack of infectious fever appears to afford strong evidence in favour of its being due to spontaneous origin, it often happens that, as the result of renewed and skilled investigation, or owing to the accidental discovery of some apparently trivial circumstance, the disease is after all found to be due to infection from some prior case. Many such instances must have been noted by the members of this Society. One which came under my own notice I will record. A woman living in a village which had long been free from enteric fever, sickened with that disease, and, after having communicated it to others, she died. The previous history of her attack had, up to a certain point, been somewhat carefully inquired into, and it was regarded as an instance of enteric fever of pythogenic origin. At a date prior to this woman's illness, which fairly represented the ordinary incubation period of enteric fever, she had started on a holiday trip, and at a certain railway junction the train she was in had been shunted on to a siding so as to bring the carriage she was in into close contact with some trucks laden with highly offensive, but otherwise ordinary farmyard manure, which was exposed to the full rays of a midday sun. The effluvia arising from this fœtid refuse was such as completely to overpower the woman; indeed, she felt quite unable to continue her journey, and at the first opportunity she quitted the train and returned to her home. She was stated never to have "got over" the smell of this manure, which clung as it were to her nostrils, and after a feeling of general malaise and discomfort, lasting from eight to ten days, she sickened with well-marked enteric fever. So far there seemed strong grounds for believing that the disease was

really brought about by the emanations in question, and that the case afforded proof that enteric fever is not invariably a self-propagating disease. It was, however, subsequently ascertained that just prior to her intended holiday trip, and also well within the incubation period of enteric fever, the woman had been to a neighbouring town, and inquiry there elicited the fact that she had stayed at a house where a fatal case of enteric fever had occurred, and that she had there been exposed to the very conditions which had led to the outbreak in the house she visited.

4. Another point which may be urged by the contagionist is, the fact that periods of incubation, known by all to be subject to certain variations in length, do in exceptional instances vary much more than is generally recognised, and in consequence constitute a source of error for which due allowance is not made. Dr. Budd, in his work on Typhoid Fever, refers to cases in which the incubation period of that disease could not have exceeded four, and even two days; indeed so rapidly did the disease in some instances supervene on exposure to infection, that it was at first looked upon as the result of some chemical poison. In at least one group of cases the proportion attacked out of those exposed to the infection was so large as to make it practically certain that the disease was in reality the result of the previous exposure, and Dr. Budd regards the unusually short period of incubation as affording evidence of a high degree of concentration of the poison received. I have myself met with instances in which the facts elicited appeared to afford evidence of exceptionally prolonged periods of incubation. In one instance I found that a young woman, residing in a hamlet which had certainly for several years been free from enteric fever, was attacked with well-marked symptoms of that disease. Nearly a month prior to her attack she had returned from a visit to a distant part of the country, where there are strong grounds for assuming that she came into contact with the infection; and it is certain that after her return home she had neither left the hamlet, nor had any person from elsewhere visited her house. In short, there must have been, according to this view, a period of incubation all but a month in length.

In this case there was, however, a peculiarity worth noting. Just within fourteen days of her return, or in other words, just about the time when the early symptoms of enteric fever ought to have manifested themselves, this young woman suffered from a sharp attack of diarrhœa, which, after lasting a day or two, left her apparently in per-

fect health, until after a further lapse of twelve days, when unmistakable symptoms of enteric fever came on. In this instance it is just possible that the attack of diarrhœa, which occurred nearly a fortnight before the ordinary symptoms of enteric fever commenced, might have been a part of the disease, and that a partial elimination of the poison was then effected. If this be so, however, we are only presented with another point of difficulty in connection with the etiology of enteric fever, which those who believe that the disease is a solely self-propagating one are entitled to claim as affording an additional argument in favour of their view.

5. And again, there are facts which are open to the interpretation that, in certain cases where strict investigation fails to elicit any information as to the possibility of recent infection, an outbreak of some acute specific fever, instead of being due to spontaneous origin, is in reality the result of infection from some prior case of the same disease which had occurred at some distant date; the specific poison having remained dormant, and having retained its vitality for a long period of time. That some "organisms", especially those of vegetable origin, do at times retain their vitality for long periods whilst in an apparently dormant state has long admitted of no doubt. And it is now further known that the "organism" found in one of the acute specific fevers affecting the lower animals, namely splenic fever, is capable of thus retaining its vitality. It exists in at least two states, in one of which it is liable to undergo rapid development; in the other it is permanent and resistant.* Some of the "spores", according to Dr. William Roberts, F.R.S., retain "their infective activity for an indefinite period in spite of all kinds of maltreatment". They can "be reduced to dust, wetted and dried repeatedly, kept in putrefying liquids for weeks, and yet at the end of four years still display an undiminished virulence".† In fact we know no reason why the infection of the various acute specific fevers should not last, under certain circumstances, for an indefinite period, much in the same way as grains of wheat have in exceptional instances lain dormant for some thousands of years and then borne fruit. With regard to scarlet fever, there are several instances on record which tend to show that its poison will remain dormant for more

* "On the Infective Process of Disease," by J. Burdon Sanderson, M.D., F.R.S., *British Medical Journal*, February 9, 1878.

† *On the Doctrine of Contagium Vivum*, by William Roberts, M.D., F.R.S.

than a year, and then act with unabated virulence. Instances have come under my own notice, in which the facts warranted the conclusion that the poison of diphtheria had been retained for months about premises in which cases of this disease had previously occurred. Dr. A. Carpenter, in a paper read before the British Medical Association in 1875, records a case in which the poison of small-pox had evidently been retained in a so-called dormant state for a long period. "He remembered a practitioner in Oban informing him of such a case occurring in Mull. A proprietor of that island ordered the removal of some old houses which had lain in a state of ruin and uninhabited for many years. Eight men were employed in digging the walls, and every one was attacked with small-pox; the germs of that disease", to use the writer's own words, "having lain in these houses all those years."

Similar evidence with regard to enteric fever has often been recorded. Dr. W. Ogle, in some of his reports on the sanitary condition of the East Herts district, refers to cases in which attacks of this disease have in successive autumns occurred in isolated dwellings, where, so far as could be ascertained by the strictest investigation, no new source of infection could have come into operation. Dr. Budd writes, "I once knew a labourer's cottage which remained vacant nearly two years, in consequence of nearly every inmate of it having contracted typhoid fever. At the end of that time it was retenanted, and three weeks had scarcely elapsed before several of the new inmates were simultaneously seized with the same fever. The cottage stood alone in a secluded spot, and there was no fever in the neighbourhood at the time of the second attack." I know a detached house which stands in large grounds in a country district, and which was occupied by a groom and his family, amongst whom enteric fever prevailed in the autumn of 1872, one case being fatal. This family continued to occupy the house for nearly two years after this occurrence, but they left it some time in 1874 in consequence of the departure of the owner of the estate on which it stood. From that date the house remained unoccupied until February 1876, when it was tenanted by new inmates; and exactly within fourteen days of these latter taking up their abode there enteric fever broke out amongst them, and a most careful inquiry led both the medical man in attendance and myself to the conclusion that the disease was not imported. I also know an isolated parsonage in which the families of two successive vicars have been attacked with enteric fever.

One more instance, which is recorded by Dr. Gilbert Child, I would refer to, because of the evidence it contains as to the communication of enteric fever, after a lapse of considerable time, by means of fomites. On the 20th of November 1873, a man arrived at a house in an Oxfordshire village, where his mother and his brother lived, bringing with him several boxes of some very filthy bedding, clothes, etc. On the 29th of the same month the brother was seized with a fatal attack of enteric fever, and on the following day the mother fell sick of the same disease. No source of infection being known, the man referred to was questioned as to his prior movements, and it transpired that in the previous September his wife had died of enteric fever at Toronto, and that he, on subsequently leaving America, had brought away with him her dirty bedding and clothing. The only other case of enteric fever which could be heard of anywhere within the district was that of a woman who was attacked on the 5th of December, and she, being also one of the man's relations, had taken to her home some of the dirty things to wash.

Instances such as these, it may well be alleged, strongly support the view that the poisons of the acute specific fevers do, under certain circumstances, retain their vitality for very long periods of time; or, in other words, that they "possess the wonderful property of passing into a state of persistent inactivity or latent vitality, in which they perform no function, but can at any moment be wakened up into active function, whenever they are brought under favourable circumstances".*

6. That the diseases under consideration are purely self-propagating may also be urged in view of the long-continued and complete immunity from them which certain isolated countries have enjoyed, and of the virulence which such diseases have exhibited when once introduced into them. Sir Thomas Watson, in his somewhat recent article in the *Nineteenth Century*, recalls the circumstance that the New World was free from small-pox prior to its discovery by Columbus in 1492, and in the same way this disease was unknown in Mexico, Iceland, and Greenland, until its importation into them, when it spread with terrible desolation among their populations. We have the same experience with regard to the long immunity which the Faroe Islands and Fiji enjoyed from measles, which disease, on being imported, spread through them with all but unparalleled

* *Lectures on the Occurrence of Organic Forms in connection with Contagious and Infective Diseases*, by J. Burdon Sanderson, M.D., F.R.S.

fatality; and a similar long-continued absence of these diseases has been noticed even when the conditions with which their spread is generally associated have been rife. Thus, conditions favouring excremental poisoning of air breathed, or of water drunk, are known to have existed for years without producing a single case of enteric fever; and yet when the disease has been imported, the violence of its spread has shown conclusively how suitable a soil for the propagation of the poison existed ready to hand.

These, besides other arguments which will doubtless present themselves to your minds, may with justice be used by the supporters of the theory that the acute specific fevers never arise independently of a prior case, and they undoubtedly constitute a somewhat formidable array, the more so when it is remembered how almost impossible it is for any one holding opposite views to state with confidence, that as regards any special case, all such sources of error can with certainty be excluded.

II. There are, however, observers, and amongst them men of great distinction, who confidently hold the opposite view, and whilst freely admitting that the diseases in question are self-propagating, yet unhesitatingly assert that some of them, at least, do at times arise independently of antecedent cases; that they are, in fact, of spontaneous origin.

As a mere matter of argument, the view they hold is by no means so easy of support as that of those who declare that these diseases are solely and exclusively self-propagating; for, whereas the latter can, in numberless instances, bring forward positive evidence in favour of their conclusions, the former have the far more difficult task of proving a negative; and the mere enumeration of all the sources of error which must be avoided before any positive conclusion can be arrived at in the matter, shows how laborious of proof the position they have taken up becomes. Those, however, who do believe in the independent origin of these poisons have important points to urge in behalf of their views.

1. Judging from analogy, they may point out how their opinions derive support from the well-known fact that "spontaneous generation of the traumatic infections" does "take place",* and that their respective contagia do arise in putrefactive wound products. As the result of some-

* Sixth Report of the Medical Officer of the Privy Council.

what recent experiments, Dr. Burdon Sanderson has found that "if the exudation of a simple peritonitis be injected fresh into the peritoneum of another animal, the disease assumes a more intense form in the second than in the first",—and if in this way the disease be communicated to several animals, and by "artificial selection the most severe case be picked out, and the exudation from that case be used for further transmission, a still more intense inflammation will be the result, until at last a virus is obtained, of which the virulence resembles that of the specific cases of malignant peritonitis in the human subject".* Here, it may be alleged, is a process in every way resembling the development by progressive stages of that which appears to be a specific poison, the starting-point being a simple inflammatory fluid exposed to a series of circumstances favourable to such development.

2. Then there is the fact that certain of the acute specific fevers are known to follow on a concurrence of certain well-known conditions, and this under circumstances which enable observers to assert that there is a total absence of anything tending to indicate the possibility of the diseases having been imported. The manner, for example, in which typhus attacks armies when they suffer from ill-feeding and over-crowding, especially when those conditions are associated with the want and misery and depressing emotions following on defeat. The outbreaks of this disease during the recent war in the East remind us forcibly of the conditions under which it is said to arise amongst large masses of ill-cared-for troops, in localities where it cannot be supposed that any previous cases of the disease have existed. The terrible havoc which typhus made, both amongst the besiegers and defenders of Sebastopol, is another case in point.

3. And, further, all careful observers must admit that not unfrequently, when cases of infectious fever break out under circumstances which appear to afford every facility for ascertaining in every detail the previous history of the persons attacked, yet no amount of the most careful and exhaustive inquiry can elicit any information justifying the assertion that the disease is due to some antecedent case. If such cases were of rare, or even of somewhat unfrequent occurrence, they might perhaps, with but little hesitation, be set aside. But no one having access to the reports of medical officers of health can fail to be struck with the number of

* *Infective Processes of Disease*, by J. Burdon Sanderson, M.D., F.R.S.

instances in which trained observers, carrying on their investigations in isolated localities free from the ordinary sources of error attaching to life in large towns, and making full allowance for all known sources of error, entirely fail to connect outbreaks of some of these diseases, especially of diphtheria and enteric fever, with any antecedent case, whether near or remote; and hence these observers feel themselves compelled to fall back upon the view that such diseases do, at times, have an independent origin.* My own failures to elicit any history of previous infection as regards cases of infectious inflammation of the throat—of diphtheria—which have occurred in isolated houses lying in districts far removed from other human habitations, have forcibly struck me, the more so as in the majority of instances those first attacked have been children of such tender years that all their movements could be ascertained with remarkable accuracy.

4. And, again, with regard to instances like those which have already been referred to in the argument on the other side of the question—instances in which a disease such as enteric fever recurs in successive years, or after a lapse of years, in houses where it has previously existed—instances which are assumed to afford evidence of the poison being retained as it were in a dormant condition; it may be alleged that the conditions favourable to the development of the poisons being present, each separate outbreak is but the result of a recurrence of the process by which a new poison is each time brought into existence.

5. And yet once more, there is an argument which cannot be lightly passed over. Those who believe that the poisons of the acute specific fevers may and do arise spontaneously have a perfect right to remind their opponents that there must have been first cases of each of the diseases under consideration, and that unless we are prepared to accept the view that the several contagia, which breed truly by a "process as regular as that by which dog breeds dog, and cat, cat", were originally the result of definite acts of creation, it must be assumed that they in some other way did arise independently of antecedent cases; and what right, they may ask, have we for asserting that a process once possible is now impossible? Dr. Budd says an argument such as this constitutes "the last refuge of the partizans of spontaneous origin"; but if it be so, it is a stronghold in itself,

* The Reports by Dr. Charles Kelly, on the Condition of the combined Sanitary District of West Sussex, contain many instructive instances of this.

and one which must at least be noticed before it is passed by.

Thus far I have endeavoured, and I trust successfully, to take neither side in the argument, and were it not for the circumstance that the "Remarks" I have so far offered would have had an appearance of incompleteness if I did not in some way indicate how I was influenced by the points brought forward, I should have preferred at once to draw to a conclusion. And if I do proceed briefly to explain the effect which some consideration of the subject has had upon my mind, it is on the understanding that I deem the matter as one still *sub judice*, and that I am unprepared to offer anything which I can look upon as a final opinion in regard to it.

There do, however, appear to me to be some grounds for doubting whether certain of these diseases, as at present met with, are exclusively self-propagating, and for raising the question whether they may not occasionally arise independently of antecedent cases. Such personal experience as has inclined me towards this view is mainly limited to investigations into the origin of outbreaks of diphtheria, and one point with which I have been specially struck is the following. In isolated districts, and in houses situated at times miles away from other habitations, and in some instances lying in lonely spots among mountain ranges, where a visit to, or a visit from, the nearest town or village would be a circumstance too important to be forgotten, I have met with instances of what appeared to me to be nothing more than a simple inflammation of the throat, at times so trivial that it has passed all but unnoticed, and yet it has led, by transmission through other persons, to cases of well-marked and severe diphtheria. The first attacks have often happened in children whose former movements could apparently be traced with the strictest accuracy; they have occurred under circumstances which did not appear to admit of previous infection; and it has been difficult to interpret their occurrence except on the supposition that in some way they have arisen independently of prior cases. With regard also to the well-marked attacks of diphtheria to which they seemed to give rise, all other sources of infection could be excluded with a degree of certainty rarely to be met with. And as to those first affected, whose cases appeared to be earliest in a series which led up to attacks of well-marked diphtheria, it has more than once happened that they were children in whom so-called "sore throat" was a common affection, and whose fauces, when examined, exhibited a loss of tissue *indicative* of former throat attacks.

I have stated that instances such as these have been met with in connection with outbreaks of diphtheria, and it may therefore be inferred that the early cases were merely mild attacks of that disease, the poison of which had been received in some unascertained method; but every one of these instances occurred in persons living miles away from the apparently very limited infected localities, which in some cases were mere hamlets, and this source of error seemed to be one which could unquestionably be excluded. And during the same investigations I have noticed the fact that over an area of some miles around the district in which genuine diphtheria was prevailing, there existed, prior to, as well as during, the diphtheria epidemic numerous instances of sore throats, which, so far as examination of the patients was concerned, in every way resembled the early cases above referred to, and yet which gave no indication of being infectious; sore throat being in fact in the surrounding district a prevailing ailment. And I have hardly been able to refrain from drawing the conclusion that conditions very similar to those under which genuine diphtheria was epidemic in a limited district existed (and had existed before genuine diphtheria was anywhere seen) over a wide area around the immediately infected locality; and that these conditions, leading to a somewhat general predisposition to simple and apparently non-infectious inflammatory sore throat, had (further probably under somewhat modified circumstances) tended at certain points to produce an affection capable of putting on the property of infectiveness, which thus led to the transmission of the disease in a distinctly communicable form to others.

If this be a correct interpretation of the circumstances related, it would appear to indicate the possible occurrence of what may perhaps be looked upon as the progressive development of the property of infectiveness. And if the contagia of the acute specific diseases do, as has been suggested by more than one observer, belong to the vegetable world, I know no grounds for refusing to believe that organisms capable of producing a minor and an uncommunicable disease in particular stages of their growth, may in other stages of their growth, or in the course of their subsequent development, become capable of producing a major disease communicable from person to person; the affair being essentially one of soil. This is not at all a question of the development of a living organism out of matter independently of antecedent life, but merely the production, by means of a process of evolution, of that which gives to an

already existing organism that property by which it becomes infective, a property which it may perhaps lose directly it is deprived of the circumstances which favoured its development, in much the same way as special characteristics may be artificially developed in higher plant life, and be as easily lost again.

ON INFECTION CONSIDERED FROM A DARWINIAN POINT OF VIEW.

By HUBERT AIRY, M.A., M.D.,
Medical Inspector to the Local Government Board.

(*Read: December 4th*, 1878.)

When among the foremost workers in the field of pathology there still remains doubt as to the intimate nature of that which causes infectious disease, it may well be supposed that the facts discovered up to the present time are insufficient to lead us to the true solution of the problem; and it might seem fitting that a student of the subject who has no new facts to produce should continue to search in silence rather than advance mere speculation. But I throw myself on your indulgence, knowing that we are all fellow-students in Nature's school, and that study may be stimulated, though it may not be directly enlightened, by speculative discussion.

It will be remembered that the subject of the nature and origin of infection was brought before this society in April of the present year by my friend Dr. Thorne Thorne. In his able paper on that subject, Dr. Thorne brought forward certain remarkable facts observed in connection with an outbreak of diphtheria, which suggested to him the striking idea of "progressive development of infectiveness".

I need hardly say that we shall look with the greatest interest to future investigations by the same author for confirmation and extension of the view which is set forth in those words. In the meantime, I will ask leave to borrow the hint those words contain, and will endeavour to indicate certain considerations which are suggested thereby.

The idea of gradual development of a specific infectious disease within the limits of a single epidemic is an idea which possesses especial importance in relation to biological opinions which prevail at the present day. Twenty years ago, the very words in which the idea is couched would perhaps have been hardly understood. In the present day, we are at least familiar with the words "development" and

"evolution", and there is no doubt that the ideas which those words convey have in recent years acquired a considerable degree of distinctness and (if I may so say) of organisation.

The genius and labours of Charles Darwin and the school of biologists of which he is the Grand Master, have brought home to most thoughtful minds the conviction that all forms of life are undergoing slow but perpetual change in co-aptation with the perpetual changes of the complex scenery of their existence. We have learnt to recognise that among the progeny of any given organism there is not perfect similarity, but there are minute deviations from a common parental likeness. Of these minute variations, some will be of a nature to favour their possessors in the race of life, and slightly improve their chance of arriving at maturity and reproducing their kind. Their progeny will inherit in some measure—and some of them, by further variation in the same direction, will in some measure extend—the same advantage. In this way, by the slow accumulation of infinitesimal steps, from generation to generation, through the long course of ages, a great change, or several contemporary but divergent changes, will be brought about in the bodily framework of the species; and new and distinct forms will thus be produced, each reproducing its like and deserving, equally with the original form from which they sprang, the rank of species.

These changes cannot be limited to the form and frame alone. They must affect also the several faculties and functions with which the various bodily members are endowed. For example, an organ which in one form can only secrete uric acid may, by slow modification, acquire in another form the faculty of secreting urea. In different plants the same organs may acquire the faculty of elaborating substances so different from one another as india-rubber and turpentine.

Thus, taking a broad view of the whole organic empire, as knit together by the tie of consanguinity, we may say that, in order fully to know a given plant or animal, and properly to appreciate the true nature and bearings of any vital process or function, we ought to study it, not by itself alone, nor only in relation to other contemporary forms, but also in relation to its historical development through the long series of ancestral forms that preceded it. The part which is played in the economy of our bodies by the liver or the heart or the blood cannot be fully understood until we have learnt by what upward steps, from what lowly origin, these organs attained their present complexity of form and function.

Now, these considerations of historical development apply primarily to organisms in their normal condition of health. But I contend that their application is not limited exclusively to that condition, but extends also, in a modified form, to the whole province of disease. For, with certain forms and faculties of certain organs there are co-related certain liabilities to disorder of form and function; and such disorder constitutes disease. Variation of form or function must carry with it a corresponding variability of the disorder to which that form or that function is liable; that is to say, there must be variability of disease.

This very feature must needs constitute one very important element in the rivalry between variant forms. The one which brings with it the least fatal appanage of disease will, so far, have the advantage over its rivals, and be the most likely to survive. There will thus be a kind of natural selection of disease, and a survival of the fittest and least fatal, except so far as the selection may be over-ruled by the greater relative advantage attaching to some rival variation which is saddled with a more fatal form of disease.

It might be thought at first that the tendency of the operation of natural selection must necessarily be to eliminate all disease—at any rate all intrinsic disease—in virtue of the better chance of survival enjoyed by individuals not subject to such disease. And this would in all probability be the case if variability in the human system were confined to its susceptibility to disease; but, considering that the human system must admit of variation at one and the same time in countless different ways, some profitable, others injurious, it is plain that the relative chance of survival of any modification of the whole system will depend upon the total value, or "algebraic sum", of all the constituent elements of that modification. And it may well be that a greater liability to disease in a given modification of the system might be more than counterbalanced by some concurrent advantage gained in another direction.

Different classes of disease will come within the operation of these causes in different ways and in different degrees; but none, as far as I can see, are by their nature exempt from liability to change. Idiopathic functional disease differs widely and essentially from the action of poisons introduced from without into the system; but both are liable to be altered by alterations in the structure or functional behaviour of the organs affected. [I only mention these diseases, which at first sight must seem remote from any connection with my subject, in order to exemplify my preliminary con-

tention, that there must be historical development of disease in general.]

A disease, in short, has a history and a pedigree; and this history and pedigree we ought to know if we are to obtain a full scientific insight into the nature and relations of the disease.

For this purpose a range of study would be required which could hardly be compassed without the devotion of a lifetime to it. A new science would have to be created,—a science of comparative pathology, parallel in great measure to the science of comparative anatomy. The comparative pathologist should first possess a full knowledge of human pathology in his own country, to enable him to institute a comparison between kindred diseases of human beings in different countries, with especial regard to those races of mankind which exhibit the lowest grade of human development. His study would next extend to the various species of quadrumana, and thence downward through the whole animal kingdom, embracing even the simplest monads on the very border-land where it is difficult to say whether they partake most of the animal or of the vegetable character. I cannot help thinking that such a study might be expected to throw a light on many of the simple elements of disease, on inflammatory and degenerative changes, on malignant growths and functional aberrations, which might ultimately guide the physician and surgeon to more certain remedies than they yet possess, and might even lead to discoveries which in their nature and importance should be comparable to the immortal discovery of Edward Jenner.

On this point I may be permitted to quote the words of one who has the very highest claim to speak with authority. Alluding to the need of scientific researches for creating an intimate knowledge as to the nature of morbid processes, and as to the physical and chemical conditions of their development, Mr. Simon says (in the Eighth Report of the Medical Officer of the Privy Council, 1865, p. 21): "The work is not of the kind which can promise either immediate returns, or such returns as are most popularly striking. But that medicine, in its most practical aspects, whether preventive or curative, needs constant aid and guidance from purely scientific researches, is a fact on which it would be superfluous to insist. Of the convictions which in this respect are universal among enlightened persons, perhaps no stronger illustration could be cited than the urgency, almost the clamour, with which such researches were recently called for in relation to the steppe-murrain in Eng-

land, and the scale on which the Royal Cattle Plague Commission ordered them. Also, in another point of view" (Mr. Simon continues), "I may refer to those researches for an illustration. Undertaken as they were in the medical interests of horned cattle, they yet elucidated some processes of disease in a way which is of interest to human pathology, and, through it, to the preventive medicine of mankind."

If it might not be thought presumptuous in me, I would venture to say that in the work of the Pathological Laboratory in Earl's Court Road, and in the researches undertaken for the Privy Council, under the advice of Mr. Simon, by Dr. Burdon Sanderson, Dr. Thudichum, Dr. Klein, and Dr. Creighton, we see already the science of comparative pathology in the germ.

I come now to consider more particularly the bearing of the Darwinian theory on a class of diseases to which I have as yet scarcely referred, but which concerns us in an especial degree,—I mean infectious diseases,—diseases which possess (among others) this remarkable property in common, that they are capable of being communicated from one person to another. Let us examine how far and in what way this property of communicability lays these diseases open to the operation of the laws of natural selection and survival of the fittest. And, first, I observe that, irrespective of any theory as to the nature of infection, there would appear to be *à priori* grounds for believing that in any disease in which a tendency to become infectious is at all possible, that tendency is likely to be developed and intensified in course of time. For if we suppose a community exposed in equal degree to a number of diseases of equal fatality, it is plain that any one of these diseases which has in any appreciable degree the property of infectiveness, will attack more persons than a non-infectious disease, and those who contract the disease in a more infectious form will be more likely to transmit it to others. In short, the more infectious the disease becomes, the more numerous will be the persons attacked by it, and the greater (*cæteris paribus*) will be its chance of survival by continued reproduction. I may remark that similar reasoning suggests itself, leading to a similar conclusion, in respect to hereditary disease, of not more than the average fatality. In either case, whether of infectious or of hereditary disease, the conditions are practically the same as those of a favoured variation in animal or vegetable life, enabling its possessors to leave more offspring after their own likeness than other less favoured forms.

I have now to consider certain theories which have been advanced to account for the phenomena of infection, and to ask which of them is most in accordance with the requirements of the law of natural selection,—though to this question I doubt if I shall be able to find an answer. For a succinct and impartial account of two theories which may be said to divide pathologists at the present day on the question of spontaneous origin of infectious disease, I would refer to the paper by Dr. Thorne Thorne to which I have already alluded. Dr. Thorne weighs the arguments advanced on the one hand in favour of the view "that no case of infectious disease can arise independently of a prior case of the same disease", and on the other, in favour of the view "that some of the diseases in question do at times arise independently of antecedent cases; that they are, in fact, of spontaneous origin"; and he finally adduces those remarkable facts concerning diphtheria which incline him (at least in respect of that disease) towards the latter view.

I think it will be admitted that the diversity of opinion here insisted upon, relates not so much to the intimate nature of infection as to the mode and manner of its exhibition. Both sides of the question, as thus opposed, admit of being sub-divided on the cross-question of the nature of that which causes infection. Those who maintain that every case of enteric fever is caused by an earlier case of enteric fever may range themselves in two camps, the one holding that the matter which carries the disease is an organism foreign to, but parasitic upon, the human body, and incapable of growth or propagation external to that body; and the other holding that the contagium is the infectious secretion of another human body. By a parallel sub-division, those who hold that a case of enteric fever may occur spontaneously will be divided into two bands, one maintaining that the contagium is a foreign organism generally parasitic on the human body, but also capable of growth and reproduction in some external nidus, and the other maintaining that the contagium is a depraved secretion of the human body, capable of being produced *de novo* by an intrinsic disease, but also possessing the property of reproducing the same disease when inhaled or imbibed into another body.

In relation to my present inquiry, as to the part played by natural selection in the development of infectious disease, the more important controversy is that which deals with the intimate nature of the infection-bearing matter.

Does a case of infectious disease arise from the reception into the body of a specific living organism? Or does it arise from the reception or formation in the body of a certain poisonous secretion? Which are we to adopt,—the theory of *Contagium Vivum*, as championed conspicuously by Dr. William Roberts, in his Address to the British Medical Association, in August 1877; or the Glandular Secretion theory of Dr. Benjamin Richardson, as enunciated in his Address to the Sanitary Congress at Leamington, in October of the same year?

It is not without the greatest diffidence that I venture to touch a subject which has been so ably handled by writers of such distinction. Each establishes his position so strongly, and maintains it with such convincing arguments, that, if either stood alone, that one would command our ready acceptance. But they stand directly opposed to one another; they cannot both be exclusively true; one must be wholly wrong, or both must be partly wrong. Pending the decision of the controversy, by those who are alone qualified to decide it, the question is still open to debate and discussion among lookers-on.

Taking first the theory of *Contagium Vivum*,—the theory that infectious disease arises from germs of some specific organism received into, and multiplying within, the human body, it is seen at once that we have here a complex problem of natural selection. We are evidently dealing with a case of parasitic adaptation (and nowhere, perhaps, do we find more evidence of adaptable variation than in cases of parasitism). We are dealing with a case which involves two interdependent variables, man and microzyme. In successive generations of mankind there must be, on the whole, a tendency towards insusceptibility of fatal infection, that is, of the particular infections that are current at the time; because those individuals who are the least susceptible of infection are, on the whole, the most likely to leave progeny; and their progeny will probably inherit, in greater or less degree, the parental immunity. I think we may reasonably suppose that, if the morbific organism itself were incapable of variation, the human race would, by natural selection, become proof against infection by that organism; unless, with susceptibility to infection, there were correlated some contingent advantages relatively more powerful than the risk of a fatal termination of the infectious disease. But if, on the other hand, the contagium is a living organism, depending for its support on human susceptibility, it must itself be capable of variation, to match the variability

of its human home and food. As the human syncrasy varies in the direction of insusceptibility, so the parasitic microzyme will, step for step, attain new virulence; and, as long as there is exposure to the risk of infection, that is, as long as man and microzyme are allowed to meet, I see no possibility of a cessation of the process.

Yet a compromise between them seems possible. For man the most serious contingency is that the disease may have a fatal termination: for the microzyme, it is not of vital importance that its human nidus should perish; and it would seem that natural selection might find its equilibrium (though continually fluctuating), and be fairly satisfied with so much variation on both sides as should leave man susceptible of infection, but not in a fatal degree. For this reason I am unable to agree with Dr. C. Bastian, when he says (in his Introductory Address on "Epidemic and Specific Contagious Disease", delivered at University College, October 2, 1871), "The fact that the general tendency in the acute specific diseases is undoubtedly towards recovery rather than towards death, speaks strongly against the hypothesis that they are dependent upon the presence of self-multiplying germs within the body. Such germs, when present, would be sure to go on increasing until they brought about the death of their host."

The same argument is used also by Dr. Richardson in his address, above mentioned.

This statement appears to me to leave out of account the possibility of such mutual modification as might be brought about by natural selection. In illustration, I may refer to the recent terrible fatality of measles, when freshly introduced among the Fiji Islanders. Their constitution had been formed in the absence of measles, and had not had the opportunity of acquiring a suitable protective modification, and thus a disease which in Europe is comparatively harmless, was able to sweep off many hundreds of an unprotected insular race.

(I remark, parenthetically, that a study of insular diseases, especially of the infectious class, would be likely to yield results possessing the same kind of interest as those which Charles Darwin obtained from a study of the forms of life inhabiting the Galapagos Islands.)

Protection against a given infection in a fatal form could, perhaps, only be certainly attained in a limited community, and after a great length of time. The most recent infections in any community appear to be the most fatal, such as in Europe, cholera and small-pox,—infections to which

the constitution of the race has not had time or opportunity to adapt itself. Of nearly all the outbreaks of pestilence which have become historical on account of their virulence and fatality, it has been recorded that they were previously unknown in the districts in which they proved so fatal. By some such phenomena, the Roman poet's imagination had been imbued with a theory of the origination of epidemic disease, which he expresses in the terse lines:—

> "Post ignem ætheriâ domo
> Subductum, macies et nova febrium
> Terris incubuit cohors."

Natural selection does not appear to forbid the hypothesis, that a race of microphytes, bred in some external abode, under appropriate conditions of temperature and aliment, might find similar, but more favourable conditions in the human body, and might at some period become parasitic therein: it might retain, or it might finally lose its aptitude for, independent existence; and thus might, perhaps, be explained and reconciled the opposite views that are held with regard to the possibility of a case of infectious disease arising independently of any antecedent case. Both views might be true: in small-pox, for example, the (assumed) parasite may have wholly lost its independent habit, and may only be capable of transmission from body to body: in diphtheria, the morbific agent may be an organism which is capable of leading a lowly and innocent existence in a well of foul water, but, translated by chance to a higher life in the body of man, can develop a malignant disease, with all the appearance of a *de novo* origination.

The bearing of the Darwinian theory on the problem of infection is fully recognised by Dr. Roberts in his Address to the British Medical Association above referred to. Speaking of the organism of splenic fever, he says, "I see no more difficulty in believing that the *Bacillus anthracis* is a sport from the *Bacillus subtilis* than in believing, as all botanists tell us, that the bitter almond is a sport from the sweet almond—the one a bland innocuous fruit, and the other containing the elements of a deadly poison".

"If contagia are organisms", he says, "they must necessarily possess the fundamental tendencies and attributes of all organised beings. Among the most important of these attributes is the capacity for 'variation' or 'sporting'. This capacity is an essential link in the theory of evolution, and Darwin brings forward strong grounds for the belief that variation in plants and animals is not the result of chance or caprice, but is the definite effect of definite (though often

quite obscure) causes." "The laws of variation", Dr. Roberts continues, "seem to apply in a curiously exact manner to many of the phenomena of contagious diseases. One of these laws is the tendency of a variation, once produced, to become permanent, and to be transmitted ever after with perfect exactness from parent to offspring; another and controlling law is the tendency of a variation, after persisting a certain time, to revert once more (under altered conditions) to the original type." This is illustrated by the way in which "cholera suddenly breaks out in some remote district in India, and spreads from that centre over half the globe. In three or four seasons the epidemic dies away and ceases altogether from among men. A few years later, it reappears and spreads again, and disappears as before." Does not this look as "if the cholera virus were an occasional sport from some Indian saprophyte, which by variation has acquired a parasitic habit, and, having run through countless generations, either dies out or reverts again to its original type? Similarly typhoid fever might be explained as due to a variation from some common saprophyte of our stagnant pools or sewers, which, under certain conditions of its own surroundings, or certain conditions within the human body, acquires a parasitic habit. Having acquired this habit, it becomes a contagious virus, which is transmitted with its new habit through a certain number of generations; but finally, these conditions ceasing, it reverts again to its original non-parasitic type. In regard to some contagia, such as small-pox and scarlet fever, it might be said that the variation was a very rare one, but also a very permanent one, with little or no tendency to reversion, while others, like erysipelas or typhoid fever, were frequent sports, with a more decided tendency to reversion to the original type. In regard to some pathogenic organisms, it might be assumed that the parent type had disappeared, and the parasitic variety only remained, just as the wild parents of many of our cultivated flowers and vegetables have disappeared, leaving behind them only their altered descendants."

A similar explanation is suggested by Dr. Thorne of the facts concerning diphtheria, which led him to the conception of "progressive development of infectiveness". He found within the limits of a brief and local outbreak cases of simple inflammation of the throat leading, by transmission through other persons, to cases of well-marked and severe diphtheria; and great care having been taken to exclude possible fallacies in the investigation, there is good reason

to accept the interpretion which he offers in the phrase above quoted. As to the possible nature of the infective matter itself, he remarks in conclusion—"If the contagia of the acute specific diseases do, as has been suggested by more than one observer, belong to the vegetable world, I know no grounds for refusing to believe that organisms capable of producing a minor and uncommunicable disease in particular stages of their growth, may in other stages of their growth, or in the course of their subsequent development, become capable of producing a major disease communicable from person to person: the affair being essentially one of soil. This is not at all a question of the development of a living organism out of matter, independently of antecedent life, but merely the production, by means of a process of evolution, of that which gives to an already existing organism that property by which it becomes infective, a property which it may perhaps lose directly it is deprived of the circumstances which favoured its development, in much the same way as special characteristics may be artificially developed in higher plant life, and be as easily lost again."

These extracts justify me in asserting, as already established, that the theory of evolution has a claim to be referred to, perhaps even invoked as umpire, in the controversy concerning the nature of infection. I feel that the question ought to be followed out in much greater detail than my time or my knowledge will permit; but as far as we have gone, I think it must be allowed that the advocates of the germ theory may boldly appeal to the theory of evolution as at least not condemning, in some respects even especially favouring their view.

Now let us see whether the glandular theory of contagious disease cannot submit itself to the same arbitrament with equal confidence and with equal success. This theory, as expounded by Dr. Benjamin Richardson, is that the matter which produces contagious disease is nothing more than the diseased secretion of one or other of the glands of the human body itself—that each particle of any one of these poisonous secretions brought into contact either with the blood of the living animal or with certain secretions of the living animal, possesses the property of turning the albuminous part of that same blood or that same secretion into substance like itself. The process of change is catalytic. It is a change by which a body is transformed by the presence of some other body which does not itself undergo change. The multiplication of the poison thus takes place through

the force of secretion of the person affected, not through the propagation of germ from germ.

That some such action does really take place in certain cases appears to be proved by Dr. Richardson's experiments in 1864-5, in which a portion of secreted fluid taken from a patient who was suffering from surgical fever following the operation of ovariotomy, had been made to produce a definite form of fever in an inferior animal by being simply brought into contact with the peritoneal surface of the animal, the phenomena being repeated through four generations of animals. It may certainly be objected that in these experiments the diseased secretion was not administered in the same way as the contagium of an infectious disease, namely, by the lungs or the stomach, but was directly applied to the membrane corresponding to that from which it was derived. It is, however, conceivable that a poison inhaled or imbibed might enter the blood unaltered and be conveyed unaltered to the gland which it had especial power to attack.

Dr. Richardson instances the poison of any venomous snake as the type of these organisms; and this illustration must go far in support of his view, if there is evidence that that poison is reproduced, that a person who is poisoned by snake-bite can, by bite or otherwise, poison another person in the same way.

However, I am not concerned at present to discuss the question on other grounds than those of the Darwinian theory. At first sight it might seem that a mere secretion could give but little scope for the operation of the law of natural selection; but it will be admitted on consideration that any matter which possesses the property—never mind how—of reproducing its like, with possible variations, is *ipso facto* subject to the law of natural selection. That variety which is most certain to reproduce its like—that shade of secretion which proves most infective—is most likely to be transmitted and survive; and thus, if the first step be granted, that a morbid secretion can in any degree induce a similar morbid action in another body, there is no reason for denying that the process might in time attain the most virulent infectiveness.

Dr. Richardson himself appears not fully to recognise the possibility of any such modification, when he says (in the Address to which I have before referred), "Each poison has a specific property, always bringing out the same disease through countless ages. From the time when man was first attacked by them, on to the present time, I have no doubt

that each of the communicable diseases has been developed from, and has depended upon, one specific poison."

The possibility of the spontaneous origin of an infectious disease is accounted for on the glandular theory by the supposition that, "under certain influences affecting glandular action the poisons may be made to originate directly through nervous impression without the necessary intervention of an infecting particle."

That the product of such idiopathic derangement of glandular function should be identical in composition and properties with the product of countless ages of transmitted infection, would be difficult to believe, having regard to the requirements of natural selection, if we supposed the communicability of the disease to depend upon the power of the morbid secretion of one body to alter and assimilate to itself by mere contact the previously healthy secretion of another body, without affecting the function of the gland-cells which produce the secretion. This appears to be Dr. Richardson's view, when, in the passage which I have quoted, he describes the action as catalytic. If this were the mode in which the infective change is brought about, there would be no reason for supposing the functional activity of the secreting gland to have undergone any alteration, the change taking place only in the secretion after it had left the gland-cells. What reason, then, would there be to expect identity between the spontaneous disease supposed to be produced by intrinsic nervous impression on the gland, and the transmitted disease which is supposed to be due to the introduction of a particle of alien secretion which may carry with it the result of ages of modification?

If, however, we might suppose that the foreign particle of morbid secretion alters the character of the secretion which harbours it, by altering the action of the gland—and if we might suppose that for each gland there is a certain disorder of function into which it is liable to fall, either under intrinsic nervous impression, or under the contact of foreign morbid secretion—then we might fairly look for identity of results from these two very dissimilar causes. Modification of secretion would necessarily imply modification of gland-function; and the morbid action of the gland might be expected to be the same, whether excited by intrinsic nervous disturbance, or by the presence of a foreign particle.

The facts which Dr. Thorne observed in an outbreak of diphtheria at Llanrhaiadr were provisionally explained by him in terms of the germ theory. A supporter of the

glandular theory, however, would not find it impossible to explain the same facts on his own hypothesis. He would point to Dr. Burdon Sanderson's experiments (which are quoted by Dr. Thorne) as showing, "that if the exudation of a simple peritonitis be injected fresh into the peritoneum of another animal, the disease assumes a more intense form in the second than in the first; that if in this way the disease be communicated to several animals, the effects will differ in different cases; that if by artificial selection the most severe case be picked out, and the exudation from that case be used for further transmission, a still more intense inflammation will be the result, until at last a virus is obtained, of which the virulence resembles that of specific cases of malignant peritonitis in the human subject". In these cases, as Dr. Sanderson says, "the direct intervention of a morbific organism is merely a matter of inference". But until the presence of such an organism is proved, the champion of the glandular theory has a right to say that the morbid action is caused by the introduced secretion, and by nothing else.

These results of artificial experiment appear to find a striking parallel in the facts of natural development of diphtheritic infection observed by Dr. Thorne, and the same explanation may be given of both.

It might, indeed, seem difficult to explain, from the point of view of natural selection, the rapidity of the development of the infective property in these cases. This, however, might depend upon the degree of variability of the gland-function. Where that variability was large, the process of selection would be proportionally rapid. It might even be suggested as conceivable that in a solitary case a morbid change in the secretion, at first innocent, might, by gradual self-infection between different parts of the same gland, attain at last to a high pitch of malignity.

But here I must check myself and draw to a close, not without a sense of groping almost in the dark. We want more facts before we can be sure of our theory; and for more facts we must look to more experiments in comparative pathology.

There are several points relating to infection to which it would be interesting to refer, such as the general immunity from a second attack, the greater prevalence of certain infections at certain seasons and in certain districts, the phenomena of incubation, and others. On all these points each of the two rival theories which I have mentioned has its own explanation to offer; but I am not aware that they

have any new significance in relation to the theory of natural selection; and I must confess myself disappointed for the present in not finding in that theory a crucial test whereby to decide the question.

Gentlemen, I have endeavoured (but I am sensible how very imperfectly) to show that the great law of evolution by natural selection operates in the realm of disease, as well as in health; that infectious diseases are doubly subject to its operation; and that, whatever theory we adopt as to the intimate nature of infection, we cannot hope to know its past history, or anticipate its tendencies in the future, unless we study it in relation to that grand principle which is paramount through all the phenomena of life.

EDMUND ALEXANDER PARKES, M.D., F.R.S.

By the death of Dr. Parkes the Society lost one of its oldest and most valued members. He was one of the original founders of the Society, and always took a deep interest in its welfare, although, by the fact of his residence at a distance from the metropolis, he was prevented from taking as active a part in its proceedings as he would otherwise have desired. For the same reason also, he felt himself precluded from taking the presidential chair when it was offered him.

He was born on March 30th, 1819, and died at Netley, on March 15th, 1876. In the earlier part of his professional life be entered the medical service of the Army, and during his stay in India, whilst attached to the 84th Regiment, he studied several of the principal diseases prevailing in our Eastern Empire. Some of his researches on these points were subsequently embodied in his publications on Asiatic and Algide Cholera, and on the Pathology of Dysentery. After his return to England he was occupied, during a very important period of his life, as a teacher of clinical medicine at University College Hospital. Later on, his services were sought by the military authorities on various matters connected with our hospitals at Scutari and in the Crimea, during the Russian War. But his name is for this Society more closely associated with the subject of hygiene, the science he has brought into system and has promoted to an extent unsurpassed by any other worker. His connection with the Army Medical School at Netley, followed shortly by the publication of his *Manual of Practical Hygiene*, have had an influence which will not end with his death.

His intellectual powers were of the highest order; his researches were always characterised with unusual patience; and his work was, in every branch, a model of honesty, earnestness, and untiring industry. With such qualities were combined a sweetness and simplicity of character which won for him the love as well as the admiration of all those with whom he was associated. It deserves notice that the intention of commemorating his connection with University College has taken the form of a museum and library for the use of students in sanitary science.

INAUGURAL ADDRESS.

Session 1879-80.

Delivered by Sir JOSEPH FAYRER, K.C.S.I., M.D., F.R.S., President.

[*November 5th*, 1879.]

Gentlemen,—My first duty is to thank you for placing me in the honourable and responsible position I now occupy as President of this Society. I must suppose, whatever be my own misgivings on the subject, that you recognise in me some personal or official fitness for the post, and it therefore only remains for me to endeavour to justify the selection you have made, though I confess I anticipate some difficulty in doing so. The duties of a Society with such aims as those of the one over which you have called on me to preside are of a very important nature; the questions it proposes to investigate are often of surpassing interest, and the influence it ought to exercise for the public good should be largely exerted and widely felt. To guide the affairs of such an institution seems to me to demand, in the person who holds the office of President, a combination of qualifications to which I can lay no claim; nevertheless, as you have been pleased to place me here, it will be my duty and my anxious desire to devote to your service such knowledge as I may have acquired during a long residence in India, where epidemic disease prevails; and for the rest, I can only crave your indulgence for such shortcomings as must occur in the case of one whose experience has been rather that of a practical physician than of a scientific epidemiologist; assuring you, at the same time, that no effort shall be wanting to promote the welfare of the Society.

On assuming this seat, I know it is my duty to inaugurate the session by a brief address on some subject cognate to the work before the Society, and I must, therefore, observe the usual routine; but I admit I approach it with some hesitation, for there is so much that might be said that I hardly know what to select as most appropriate to the occasion. I trust, however, that I shall not greatly err in selecting a subject connected with the progress of epidemiology in that great country which contributes to the British Empire 250,000,000

inhabitants, composed of many races; and territory as large as all Europe, excluding Russia—including every climate, from that of the arctic to the torrid zone; and which is regarded by many as the birthplace and permanent home of the most terrible epidemic disease to which the human race is liable.

We are now entering on our thirtieth session, and, I trust, we have reason to believe that our progress is satisfactory, and our work such as will prove that the Society is fulfilling the main object for which it was founded. Though not one of the largest, it is certainly not one of the least active or important among the medical societies of the metropolis, whilst the cosmopolitan range of subjects embraced within the scope of its inquiry renders its proceedings of far more than mere local interest. The papers read and subjects discussed during previous years vindicate its title to a prominent place among institutions which have for their object the promotion of some of the best interests of mankind; and I can only express a hope that the session now commencing will not fall short, in this respect, of its predecessors.

As a comparatively new member of the Society, I have as yet had but little experience of its mode of working, and few opportunities of taking much part in its discussions, but I have learned enough of its objects and proceedings to know that they merit the best efforts of the members for its support, and also for the elucidation of the important (and, in some cases, still undetermined) questions that come before it, and which so deeply concern the welfare of man and the prosperity of nations—problems relating to the genesis, diffusion, and prophylaxis of disease which, in the epidemic form, still so often proves the scourge of communities and the enigma of science. Such are the subjects it is our duty to investigate, and we hope by degrees in some measure to unravel. Nor need we doubt that in time much of what is now obscure will be made clear. When we think of what has been done but recently—how much has been learned by careful observation and generalisation, how beneficial has been the result of the knowledge as applied to sanitary work, and in the diminution it has effected in the death-rate; and when we contrast our present knowledge of idiopathic fevers and other diseases, the laws of contagion, and the natural history of entozoa, with that of but a few years ago, we may reasonably hope that the work of such a Society as this will be prolific of good results, aided, as I trust it always will be, by the experience of men who have studied disease, not only by the bedside of metropolitan hospitals, but in all climates,

in every quarter of the globe, in the army, navy, colonies, and in our Indian empire (which may be regarded as the head-quarters, not only of epidemic, but also of some special form of indigenous or endemic disease). Regarding it from this point of view, we naturally desire to have a large accession of members on our rolls, and to see a greater share of the interest that is so freely bestowed on the pathology and therapeutics of special forms of disease accorded to epidemiology, to disease in the aggregate, or as it affects the masses of the population, when, in obedience to laws with which we are as yet but imperfectly acquainted, it moves in and from city to city, over rivers ard across seas, from province to province, from continent to continent, until at length it disappears apparently as mysteriously as it arose, though, no doubt, in conformity with constant and definite laws. No one can have watched the rise, progress, and decline of an epidemic of cholera or of fever, such as the dengue that last swept over India in 1872, with its apparent caprices of invasion and subsidence, activity, and decline, without feeling how much he has still to learn before he can comprehend the import or reason of it all, and how essential it is that we should study, with all the aids that science can bring to bear on the inquiry, the complex and recondite agencies or forces that control the origin and movement of epidemic disease; patiently studying its various phases and phenomena, carefully substantiating facts, not allowing ourselves to be unduly influenced by the bias of preconceived theories, and endeavouring to record these facts *as they are*, and not as we think *they might be*, for thus shall we gradually accumulate reliable data from which to generalise, and on which principles may be based and laws established. Nor is such caution superfluous, as I think will be admitted by those persons who have had to deal with sanitary reports.

The *raison d'être* of this Society is the investigation and development of our knowledge of disease in motion. It involves much, for any disease which spreads, whether among the people of a house, a ship, a village, a city, a province, or a continent, is an epidemic, and comes within the scope of our inquiry. Dysentery and malarious fevers are typically *endemic* diseases, but in India they may and do at times assume a dangerously epidemic character. But it is not meant that our inquiries should be restricted to mere epidemicity alone; we cannot advantageously study one phase of the natural history of disease and exclude others. There is so much in etiology, semeiology, and pathology, both

human and comparative, concerning our department of research, that we may not ignore the means by which we gain the most important of all information to the epidemiologist—namely, the means of discriminating one form of disease from another. Therefore, though our proceedings will naturally refer mainly to epidemic disease, we shall thankfully receive and carefully consider all information that may tend in any way to throw light on the casual relations and on the influences exercised by climate, season, locality, food, and occupation, on the genesis and dissemination of all diseases, not excluding those of the lower animals, or even of plant life. I might illustrate this by referring to the importance of discriminating between the different forms of fever occuring in India. It is well known that the greatest proportion of mortality in India is ascribed to fevers. The highest death-rate* was registered in Bombay, where it equalled 20.82 per 1000; in Madras, the deaths of 469,241 persons gave a death-rate of 10.08 per 1000. Both here and at Bombay, however, the mortality recorded was greatly aggravated by famine. The statistics of "fevers" in all years are admitted to be most inaccurate, but the deaths registered under this head in the different provinces during the year 1877 were as follows:—

Bengal	711,037
North-west Provinces ...	574,722
Punjab	219,281
Central Provinces	131,123
Berar	34,453
British Burmah	261,001
Madras	469,241
Bombay	336,865
Assam	18,725
	2,756,448

In 1878 there died in Bengal, of diseases classed as fevers, 742,887 persons (419,294 males, 323,593 females). The total death-rate from these diseases was 12.38 per 1,000, against 7.58 per 1,000 average on the previous five years. Part of this may be attributed to better registration.

In 1877, of a population of 181,929,889, in nine provinces of India, there died of fevers 2,521,438; whilst of cholera 635,977 died. In 1874 only 81,266 died.

* *Vide* Report of Sanitary Commission of the Government of India for 1877.

In his report for 1878 the sanitary commissioner (Dr. Little) of the Hyderabad assigned district of 2,756,448 inhabitants shows that there was exceptional mortality during that year. In a population of about 2,186,988, the death-rate was 81½ per 1,000 in 1878, against 32.1 per 1,000 in the previous year. The total deaths in the year were 178,404, and the causes were as follows, fever being the most prominent:—

Death causes.	Total deaths.	Deaths per 1000.
Cholera ...	34,306	15.6
Small-pox ...	5,850	2.7
Fevers ...	85,260	38.9
Bowel complaints	27,577	12.6
Injuries ...	1,147	.5
All other causes	24,264	11.1
All causes ...	178,404	

This great mortality is probably partly attributable to the indirect influence of the famine, which deprived the province of much of its most wholesome food, by causing exportation of grain to the famine districts. The population, to a great extent, thus subsisted on an inferior or deteriorated kind of grain. There were other local climatic causes, but to these I need not allude. I refer to the subject as an evidence of the great mortality of fevers, as compared with other diseases.

There is no doubt that under the heading Fever many deaths from other causes are recorded, and we may probably refer a large proportion of them to diseases of an inflammatory character affecting the thoracic or other viscera, or to complications involving inflammatory action elsewhere. In a vast country like India, where the population is so extensive, and the means of registration of necessity limited, often not under medical supervision at all, it is not to be expected that greater accuracy can be ensured; but, were it possible to discriminate among the various forms of disease returned as causing the mortality by fever, we should have a very different result from the present one. One can hardly refer to this subject without expressing admiration at the great progress that has been made of late years in registration under the direction of the sanitary authorities of India, as well as confidence that it will continue to improve, and render the statistics more valuable even than they are now. Of course, where the registration of death is not subject to medical definition, discrimination between the various forms of fever or other death-causes could hardly be expected; and there-

fore the example I have just given is hardly so good an illustration of what I refer to as typhoid, for which we have accurate medical statistics of our European troops in India. It is within my recollection that attention was first called to the existence of this form of fever in India, and yet there can be, I suppose, no doubt that it has always been there. It soon became generally recognised as a new discovery in India, and people wondered how it had escaped observation hitherto, whilst some perchance regarded it as a new disease. But it was just this power of discriminating observation, so rare and so valuable, that had been wanting; it was this, exercised by Budd, Jenner, Murchison, and others after them, established a new era in the nosology of fevers in England; and which, a little later, in India, discriminated between certain forms of remittent and enteric (*i. e.*, between malarious and specific) fevers, and will, I hope, ere long further discriminate and rearrange the nosology of fevers in India and the tropics.

Now, typhoid or enteric fever is an important cause of mortality among our young European soldiers in India; and it raises or suggests questions of great importance in regard to them—such, for example, as the right age, time, and seasons for sending them to India; to say nothing of the hygienic questions as to sanitary measures arising out of its causal relations. The Sanitary Commissioner's Report of 1877 says that out of 233 cases of typhoid, 92, or 39 per cent., proved fatal; the admission rate being 4.1 per 1000 of strength. It moreover appears that 2.45 per cent. occurred at or under twenty-four years of age, 1.55 at twenty-five to twenty-nine, 0.99 at thirty to thirty-four, and few or none above that age; showing that the disease tells most severely on the younger men—in this respect resembling typhoid in England. Again, Bryden, in his Report of the Statistical History of the European Army in India up to 1876 (published 1878), says: "It has no geography; and it is a matter of popular observation that no regiment or battery escapes enteric fever in the first year, whatever cantonment of India may be selected." "Out of seventy-three bodies of men, two regiments and seven batteries only returned no case of enteric fever in the first year." And he gives the following analysis of 368 deaths that occurred between 1823 and 1876:—

Ages.	Total deaths.
24 and under	255
25 to 29	90
30 to 34	17
35 to 39	4
40 and upwards	2

Seventy-five of these deaths occurred within three years after landing in India, and 94 per cent. of the total were among men under thirty years of age. In a memorandum received only a few days ago Bryden says, out of 132 deaths from enteric fever in 1878, 90 occurred in men who had been under twenty-two months in India. All this shows that youth and the first year of service in India are the great predisposing causes.

Now, is this the same disease as that which might be contracted in London, Dublin, Windsor, or elsewhere in a town or barrack, from a watercloset, drain, sewer, well, or it may be, from a milk-can? I have little doubt that very frequently it is exactly identical; but I believe also that perhaps as frequently, or more so, it is not; and this, I believe, not in regard to young European soldiers only, but of the whole population of India. In short, I am, and long have been, of opinion that a form of fever exactly like European typhoid, except in its etiology, exists in India and other hot and malarious countries; and that it is due to climatic causes, not to filth or specific causes such as give rise to it in England and elsewhere, and recent reports from India and other parts of the world seem to show that this view is gaining ground. I am indebted to Surgeon-Major A. Clarke for the following note:—

"Typhoid fever has prevailed very extensively in Natal and Zululand during the war. Since Jan. 1st, 1879, to May 31st, no less than 267 admissions for enteric fever have been recorded. Many of these occurred in healthy camps on ground previously unoccupied (virgin soil), and in bodies of picked men. The water-supply, as a rule, good; no sewers or drains; conservancy, dry earth or trench, and carefully attended to. The troops of Crealock's Division, which were encamped near the coast, and in close proximity to marshy ground, where the natives suffer severely from remittent fevers, had more admissions from enteric than the Second Division, which was inland and generally at higher elevations. The medical officers differed as to this fever, several maintaining it was not typhoid as commonly understood in England, but bilious remittent with typhoid symptoms, as seen in India; others nailed their colours to the mast that it was genuine enteric. In support of the former, a medical officer just home reports that numerous cases entered as enteric were discharged, and at their duty five or six days afterwards. Surgeon-General Woolfreyes describes this fever as 'typho-malarial'. He says: 'It is not a fatal fever, but it causes great prostration, and a

change to England is absolutely necessary. I am of opinion that it is climatic, the true autumnal fever. It, as a rule, commences with sore-throat, a peculiarity; the rose spots are invariably present, and in fatal cases the lesions of Peyer's patches are well marked.' At the time that enteric was reported so common in the First Division, jaundice also prevailed very extensively, but was unknown in the Second Division. Enteric has also been reported as causing much sickness amongst the troops in Afghanistan. Here, again, camps were often pitched on virgin soil, though the water-supply was far from satisfactory, and dead camels so constantly polluted the streams. It prevailed with cholera and severe remittent fevers. Altitude made no difference, cases being admitted in camps several thousand feet above sea-level. Enteric cases are reported from nearly every station in the Bengal Presidency, some, such as Cambellpore in the Punjab, where the 'filth' element is at a minimum. At this station there is no large bazaar or city in the vicinity; the soil is arid, sandy, and very dry; the rainfall exceedingly small; water-supply good, and very carefully filtered and attended to; the dry earth conservancy is carried out to perfection; the milk danger is almost nil; yet enteric occurs, and amongst the troops who drink hardly any milk, while the women and children who probably drink a good deal of bazaar milk have so far escaped. These facts seem to show that enteric fever cannot be always ascribed to a 'filth' cause, but that climate or what is embraced in that unknown word 'malaria', may give rise to it, as it undoubtedly does to intermittents, remittents, and possibly cholera."

Be it clearly understood, however, that I do not for a moment dispute the existence of genuine filth typhoid in India. The official returns, which cannot be gainsaid, and my own experience, alike leave me in no doubt about it. But I do believe that many cases now recorded, and rightly recorded, as typhoid in India, are not caused by the same specific agency as that which gives rise to typhoid here, and also in India, where the necessary conditions are present. That there are, in fact, two, perhaps more, forms of typhoid, or perhaps, I should say, different causes for a disease presenting the same phenomena, one being specific, the other climatic; if so, it is obviously very desirable to discriminate between them—not merely as a matter of nosological or pathological interest, but in regard to the hygienic measures that are necessary in either case. This question is of great interest to epidemiologists, and is just one of those subjects which should come within the scope of our

inquiry. A remarkable instance occurred near London lately, where an epidemic of typhoid was traced to its origin along the track of a particular water-supply by a distinguished member of our Society. Our lamented colleague, Murchison, also traced a similar outbreak to contamination of milk by polluted water. The explanations of these outbreaks were exactly in accordance with those views, no doubt rightly held in Europe. Typhoid in India, however, would not always be similarly explained.

Questions of this nature are, I think, such as might be advantageously discussed here. I am aware that great differences of opinion exist among those whose views command the highest respect. Now, to compare ideas, to discuss, and, it may be, to reconcile differences, to clear up doubts for ourselves and others, to confirm or refute, seems to me to be our legitimate work. Our views on a particular subject may be unsettled; they may be influenced by preconceived theory, and altogether far from confirmed. One can understand that a cautious and reflecting mind may desire more evidence before accepting either view presented as the correct one, or before admitting that it has been convinced. In regard to some questions of etiology, such, I believe, is far from an uncommon frame of mind. The consideration of such vexed questions is most desirable. Let us hope that free expression of opinion, and temperate discussion of views, however opposite, will here be attended by good results.

The late Mr. Bedford, whose comparatively early death was deplored by the Bengal Medical Service, of which he was a brilliant ornament, said in a letter to the President of the Society in the year 1850: "Epidemic diffusion is the most important medical question of the day, and can only be studied through systematic and parallel observations conducted by a number of intelligent men working under one head. India, from its climatic peculiarities, and the nature of the service, offers, I hesitate not to affirm, the most magnificent field in the world for carrying out such a series of connected observations as may tend to elucidate the laws of the most terrible disease which flesh is heir to, and it will redound to her eternal honour to be in advance of such inquiries." Whether these aspirations have in any way been fulfilled will appear from what I have to say; it will, I think, be seen that India has contributed in no small degree towards the attainment of the end so much desired; and that we owe much to the labours of those distinguished men whose writings, published in monographs, in reports,

and official returns, afford ample proof how earnestly they have laboured to advance our knowledge of disease. The names of Jameson, Scott, Annesley, Twining, Martin, Mackinnon, Morehead in earlier times; of Goodeve, Chevers, Mouat, Bedford, Ewart, Bryden, Murray (our late distinguished President), the Cunninghams, Payne, Lewis, Macpherson, Cornish, Macnamara, Hewlatt, Townsend, Coates, Planck, and many others subsequently, to say nothing of the numerous valuable medico-topographical reports by the medical service generally, represent a literature of epidemiology that is unsurpassed in richness of its material, and affords evidence that the subject has received and is receiving elucidation, that bids fair in time to throw such a flood of light on the natural history of epidemics as will reduce our knowledge to a state of exactitude hitherto unknown. They have, it is true, taught us but little of the nature of the causes of epidemics such as cholera, or of malarious diseases, but they show what may be expected in regard to them, when and how they will appear, and how sanitary work may be made most effectually to avert, prevent, control, mitigate, or remove them; nor is it too much to say that we hope, if not to banish or stamp out, at all events still further to mitigate their ravages. This, I think, is warranted by experience, for certainly the past twenty years have witnessed great progress, and we already see that not only is the value of life increasing, but that the virulence of, and mortality caused by, epidemic disease are being controlled, whilst the vexatious and purposeless restrictions and restraints of quarantine are diminished if not altogether removed, in our dominions at least. Science that has enabled us to reduce the death-rate among our troops from 17.9 to 8.56 per 1,000 in Europe, and from 69. to 17.62 per 1,000 in India, speaks for itself, and were there no other result, this alone is a triumph such as has been achieved by no other department of knowledge. Pray do not suppose that I claim all this for Indian workers. We all know that these great steps in social and sanitary progress began here, but I do say that the torch then lighted has since been worthily and firmly borne in Indian hands.

This Society, young as it is, can remember the commencement of systematic sanitary work in the East, and may claim some share in the origination of the good work; for among its earlier members were some who advocated the study of epidemiology and hygiene in India. I cannot now stay to dwell on this, but I may say briefly, in regard to its progress and work, that until the sanitary department was formed, less than twenty years ago, comparative, I might also say com-

plete, ignorance on the subject of epidemics, and of the diseases that prevailed among the people, existed. An epidemic might carry off thousands, but we knew not where it commenced, where it ended, or what area it occupied. Now, thanks to the continued and careful statistics, we know all that and more with fair accuracy, and are gradually collecting facts which make the study of epidemiology possible. Before organised sanitary work in India began our knowledge of the general population—nay, even of the European troops and prisoners—was most imperfect. Now, thanks to that department, and especially to Bryden, whose name cannot be too prominently mentioned in connection with the subject, we have in his most elaborate and valuable statistical reports, facts and figures, as well as deductions, which deserve the closest attention. We have, in short, the most complete details of sickness and mortality in all classes over the whole of India. It is impossible, however much anyone may differ from his conclusions, not to recognise the great value of his work, for these reports contain a vast and continued array of authenticated facts, which will serve as a mine of information to epidemiologists; and there is every reason for believing that it is but an earnest of more, for if, as Bedford said, the circumstances of India were such as to favour the acquisition of knowledge in 1850, when the precise conditions of life of the population generally, and even of our troops and prisoners, were imperfectly known, what must it be now, when a system of observation, carried out by a body of trained observers under a head, such as he contemplated, is in full and daily improving operation? One can only wish that, considering the magnitude of the work, it were more extensive still, and that observations, already of the greatest value, could be rendered still more so by being concentrated on certain limited areas, so as to enable the inquiry to extend to details with a precision that at present can hardly be practicable. The results of epidemiological knowledge and sanitary work are seen in the effect already produced in reducing the mortality from cholera and other epidemics, and from malarial fevers. For instance, among our European troops, the circumstances of which are well known, there has been the following alteration in the general death-rate:—

1861 to 1865	... ...	9.02 per 1000.
1865 to 1870	... ...	6.98 „
1870 to 1875	... ...	3.23 „
1875 to 1876	... ...	2.3 „
1876 to 1877	... ...	.84 „

And it can be shown from the same source (Bryden) that there is a similar reduction in the death-rate among native troops; and that during the great cholera epidemic that prevailed in 1876, the death-rate among the civil population being 12.12 per 1,000 (it was the famine year), that of the European troops was 1.75, and of the native army 2.2 per 1,000. Also that in the death-rate of that great scourge of India—fever—there has been an equally happy result, as shown by the death-rate of a period of nine years—from 1868 to 1877—in the Bengal and N.W. Provinces gaols, as against a mean, in nine years ending 1867, of 22.41.*

	Per 1000.		Per 1000.
1859	13.76	1868	2.84
1860	49.19	1869	4.57
1861	38.14	1870	6.20
1862	30.81	1871	5.81
1863	25.44	1872	1.92
1864	29.96	1873	1.56
1865	7.65	1874	2.67
1866	5.23	1875	3.50
1867	3.12	1876	1.26
Mean ...	22.41	Mean ..	3.29

In India, as elsewhere, the purpose of epidemiological study is to observe accurately, and to interpret the import of the facts—*i. e.*, if possible, to elucidate the laws of which they are the expression,—and thus to form a scientific basis on which to direct sanitary work, which itself is the practical outcome of such observation, and concerns itself but little with theories. Those who have to do with it know how difficult it sometimes is to obtain reliable observations, such is the difficulty of excluding the bias of preconceived theory of the nature of the subject under investigation. Our Society, however, must hold the balance, and deal with theories as well as facts. No doubt the explanations sometimes are conflicting. Happily, in the practical mode of dealing with the question, there is not such conflict, and it is remarkable how little different is the action of those who hold opposite opinions on the causation of disease. As to the different views that are held in regard to fundamental questions regarding the genesis and diffusion of disease, let us hope that we may, perhaps, here contribute something towards their adjustment.

In illustration of the state and progress of epidemiology in India I might select the history of any of the great epi-

* Bryden's *Report* (1876), p. 157.

demics that have occurred of late years, but it would be impossible, in the short space of time at my disposal, to do this completely. I shall, therefore, confine myself to a few remarks on cholera, as it, though far from being the most destructive, is the epidemic to which most interest attaches. No disease has been more carefully studied, and the means of doing so exist nowhere so abundantly as in India, for whether in the sporadic, endemic, or epidemic form, it is seldom absent from some part or other of the empire. In the so-called endemic area in Bengal the opportunity of studying it is never altogether absent, whether it be sporadically or moving as an epidemic within the limits of the endemic area (its permanent home), or in the periodic outbursts, when it passes over the continent generally, in what seems a capricious manner, but according to what really, as shown by Bryden, is the operation of laws that are now beginning to be well understood. The mode of invasion and diffusion, the rise and decline, the influence of locality, season, meteorology, conditions of soil, air, water, food, are all now closely studied, and to them various degrees of importance as factors have been assigned by different observers; and from the statistics, conclusions have been drawn by Bryden and others that point with considerable force to the nature and the constancy of certain laws governing the progress of the disease; and albeit they have not as yet resulted in any very striking discovery, they seem to show that climatic and local phenomena form an important basis of a useful knowledge of the etiology of cholera, whilst the researches of Lewis and Cunningham point to certain conditions of soil as determining its production and development, as being most in accordance with the phenomena of its seasonal prevalence in the endemic area. But their prolonged and careful researches have as yet thrown no new light on the nature of the cause itself. Though it is only recently that this numerical method of dealing with epidemics has obtained in India, it is to be remembered that much valuable information has been recorded in times past in the writings of those to whom I have already referred and others, and in the records of the Medical Boards of the three presidencies, and reports of the sanitary commissioners. Indeed, since the advent of the Portuguese in the sixteenth century, it has been described so graphically that one can hardly doubt the identity of the disease mentioned in early writings with that of the present day; in fact, it possesses as well defined a history as any other event of the past, and seems to have manifested the same phenomena, and to have been subjected to the same laws then as at present.

The theory that cholera is purely of Indian origin, and that, wherever it may appear, it is to be traced back ultimately to the delta of the Ganges, is disputed by some who see in history evidence that it has long existed in other parts of the world, and that it was described by the earliest writers—Sanscrit, Greek, and Arabian. I may here just remark that the name "haida", or "haiza", used by Rhazes nearly a thousand years ago in describing the symptoms of cholera is the same as that applied to it now by every Hindustanee-speaking native of India. As to its presence in India from earlier periods, we have descriptions by Correa, d'Orta, Bontius, and others, beginning from 1503. An epidemic of it in and about Goa in 1543, for example, is described by d'Orta, giving all the characteristics that distinguish an epidemic now. He calls it "monyxy" and also "haiza". It is described by a continuous chain of writers, as occurring in various parts of India, in the interior as well as on the coast, up to the seventeenth century, when, after being quiescent during the latter part of the eighteenth and early part of the nineteenth century, it broke out with great virulence in Bengal, and has remained there ever since, in what Bryden calls its endemic area, whence it spreads according to certain laws, which are being worked out with admirable patience and intelligence by this distinguished statistician. I cannot now trace the history of cholera in either East or West, nor refer even to the numerous authors who have described it. For full particulars on this subject I refer you to Inspector-General Dr. J. Macpherson's learned work, *The Annals of Cholera*.

I need hardly insist that cholera is not a new disease, or that it did not, as by some supposed, make its first appearance as an epidemic in Jepore, in Bengal, in 1813; though no doubt since that period it has been more closely investigated and described. All seems to show that it is the same now as formerly, and that though we have gained much knowledge of its natural history of late years, yet we are as ignorant as our predecessors of its real nature. We have, thanks to sanitary measures, disarmed it of some of its terrors,* and have diminished the mortality it caused; but

* And yet how little we can do. During this year, 1879, there were among the European troops in Bengal the following admissions for, and deaths from, cholera :—

	Admissions.	Deaths.
Officers ...	... 18	... 13
Men	... 803	... 605
Women ...	... 11	... 8
Children ...	... 14	... 12
Total ...	... 846	638

as to treatment we have gained but little, though the empiricism of to-day is more scientific than it was in former days. We do not now burn our patients on the soles of the feet, tie ligatures round their limbs, or have recourse to other senseless barbarities; for we find that simpler and more rational methods are of greater avail, more or less according to the period of the epidemic and the promptitude with which the remedies are applied. But we have learned that local causes have a potent influence, and that cleanliness, good air, pure water, and free ventilation are all powerful opponents of cholera; that we can predict its appearance and avoid it in certain places; and that it is not to be controlled by quarantine or sanitary cordons. And, from the earnestness and intelligence with which the subject of its etiology is pursued, it is not improbable that sooner or later it, too, will be made out. We shall then be in a position to say not only what it does or will do, but what it is. Meanwhile we must go on observing and investigating. It is satisfactory to know that we are daily learning, practically, better how to deal with it, and how to modify its cyclical intensity and avoid its ravages. For my own part, until I know something more of the nature of the cause—be it a material poison, aërial or telluric, a miasm, or a dynamic agency that so perturbs the vital energy, I cannot see my way to formulate a definite theory either of the nature of its origin or the method of its diffusion. I find the highest authorities at complete variance on the subject, and reposing faith in theories diametrically opposed to each other. Some explain all the phenomena by contagion—*i. e.*, communicability in some way of a materies or germ from one person to another. Cholera, they say, is the result of infection by a poison derived from the intestines, and water or air, but especially water, is the channel through, by, or in which the infective material is generated and conveyed. These arguments are supported by an abundant array of facts, and have been maintained by men whose very names carry conviction. Others reject altogether this explanation; they insist that local influences are all-important, and deny that the spread of cholera is due to human intercourse, that there is any poison transmitted by the excreta, or that the disease is in any way communicable from one person to another. They admit the existence of a poison of some sort—a miasm or an influence, though of its nature they are ignorant. It is a subtle thing that travels in certain directions in obedience to certain laws, is influenced by atmospheric and telluric conditions, and where it goes and finds

certain local conditions, and the people prepared by them to submit to it, there cholera will prevail. They deny the efficacy of any enteric or specific poison in the water to produce it, though they attach the greatest importance to the purity of water from all organic contamination, impure water being one of the local conditions which, if added to crowding, filth, or other sanitary conditions and want of proper ventilation, is that, of all others, which favours cholera. Such are the principal theories of the disease, as they are supported by fact and argument, which appear convincing. Europeans, Americans, and some authorities in India, maintain the contagious view, whilst high authorities in India adhere to the opposite theory, and declare roundly that the facts of cholera, in India at least, are altogether opposed to the contagion theory.

Dr. Coates, in his last report on Bengal for 1878, says:—"Much has been written, and much discussion has taken place during the year, regarding the connection between cholera and foul water. We have not yet discovered the poison of cholera, nor even if there be one. To limit the cause of cholera to one factor, and its entrance into the system to one mode only, would be very unwise. Short of demonstration, the objections against such inferences must be great. Notwithstanding this, I confess that every year's observation, in going from district to district, in hearing the various opinions of the local officers on the subject, and in reflecting on both, my conviction has become stronger and stronger that there is a connection between impure water and cholera, and the one—in Bengal at least—is the chief (I would not make it absolute or conclusive) exciting cause of the other. Wherever the drinking-water has been most perfectly kept free from impurities, and especially local ones, there cholera has prevailed the least."

I have no intention now of entering into the controversy, and have merely referred to the main points of difference that exist among those who, having watched the disease closely and critically, are equally entitled to be heard. So far as I can see, no theory yet propounded meets all the difficulties, or explains all the phenomena manifested by cholera, at least in India. There is much in each that commends it to acceptance, though each is beset by difficulties. So far as I can judge, the direct contagion theory has now few supporters anywhere, for few believe that mere contact with the sick is attended with danger, and many believe the same with regard to the excreta, the enteric contamination of water—the water theory, in fact. This is not the time or

occasion on which to offer an opinion on the matter; but I must say that, as far as my experience goes (and it has been pretty extensive), it inclines me to look for the explanation in wider and more general causes than those of infected water only. I feel unable to declare myself an unqualified adherent of any theory yet propounded; the time, I venture to think, for dogmatizing has not yet come. We may, I believe, neither accept nor reject altogether the contagion theory. I doubt if anyone can feel so confident as to say that the diffusion of cholera is altogether independent of contagion through some sort of human intercourse, or that it is not in this manner imported or conveyed to other localities. It is possible this may play a part, though a subordinate one, in the transmission of the disease; and yet I apprehend one must look for other and more general causes to account for all the phenomena. It must be borne in mind also that there may be influences and forces at work of which we know nothing, and that cholera may have different modes of working in India than in Europe. It is so with other epidemics. Yellow fever is subject to certain laws within certain parallels of latitude, or rather isothermal lines, but beyond these it is extinguished. Scarlatina can barely exist in India; it dies almost as soon as born. Cholera thrives for a time in other climates and countries, but perhaps under other conditions than those of India, and does not endure. That may be signally true of it in India which is only partially so in other climates, and the laws which regulate its diffusion may not be—probably are not—alike in all countries where it has occurred. We are hardly in a position yet to act on any theory that has reference only to its origin. Strangely enough, the two most opposed in nature as working hypotheses, seem to have very little influence on the mode in which they are practically applied to sanitary work.

I would quote some words of a former president, Dr. B. G. Babington, which are not inappropriate:—"In the infancy of geology, first studied in this country, many phenomena observed in the arrangement of the earth's crust, as it is found in this island, were supposed to furnish fixed laws, and thus gave rise among our philosophers to divers ingenious generalisations. But when these same philosophers had, from the establishment of universal peace, the opportunity of taking a wider range and of studying the earth's structure, not in this country alone, but over the whole surface of the globe, they discovered in many instances that what they had supposed to be general laws were after all only exceptional cases. We require, therefore, in a study of

[illegible]

[illegible] *

[illegible] This is [illegible] the theory which asserts that [illegible] even to the [illegible] these theories cannot be [illegible] is false. If cholera is spread [illegible] the theory which shows it to be [illegible]. If cholera is spread solely as an aerial miasm, then the theory which recognises only the effects of human intercourse is untenable. The most that is admitted by the advocates of the human theory is, that the subjection of cholera to meteorological agencies is absolute, and that these influences can and do operate so as to do away altogether with the effects of the poison, although imported. They do not recognise the entity minus the human being. The antagonistic theory, while holding, as opposed to demonstrable fact, the statement that cholera moves only by human agency, may, if necessary, be extended in its scope so as to embrace the other: that is to say, the inquiry is left open as to whether or not the cholera entity, after being aërially distributed, may be subsequently propagated or spread by man. In the theory which connects cholera solely with man there is no such extensibility. Human intercourse must explain every fact of spread and propagation, and nothing is left to the play of natural agencies. Human intercourse, giving the widest scope to the signification of the term, cannot pretend to account for any fundamental phenomenon displayed during the progress of epidemic cholera; and, therefore, I assert the theory to be radically untrue as applied to the behaviour of cholera in

* Address to Epidemiological Society, by Dr. B. G. Babington, December, 1850.

India. I do not say that the above statement will hold true all over the world; and, even as applied to India, the theory does not preclude the possibility that cholera may be conveyed by the human being. Naturally, the recoil is to the opposite extreme. The bold statement that cholera is never spread over an area unless human agency intervene, is apt to be met by the equally dogmatic statement that cholera is as pure a miasm as malaria, and as little amenable to the control of man."

I would, in passing, just remark on some curious and interesting facts regarding the introduction of epidemic disease into certain localities where the exact conditions of the people and the visitors were known. The nature of the miasm or influence is altogether unknown, but it can hardly be doubted that something was imported. Doubtless, as science progresses, epidemiology will give a simple and rational explanation of such things, and it seems to me probable that the diffusion of epidemic diseases may have to be explained on some law of which the following instances are only varieties of expression.

St. Kilda is one of the outer Hebrides, a small island north-west of Scotland. In a work on this island by Mr. George Seton, M.A.Oxon., mention is made of a peculiar form of epidemic influenza known as the "boat cough" or "stranger's cough", having all the symptoms of influenza. By this the natives are almost always attacked shortly after the arrival of a vessel from the outer Hebrides. They allege that the disease is most severe when the visitors come from Harris, and that they suffer less when the vessel hails from Glasgow or the distant ports. The malady first attacks those persons who have come most closely into contact with the strangers, and then extends itself over the whole community. By some it is attributed to the inhabitants exposing themselves to cold by rushing into the water to assist the strangers in landing, but this is not a probable explanation. Others allege that it is due to easterly winds, but the ships generally arrive with a westerly wind. By some it has been remarked that the cough is a yearly epidemic, but Mr. Morgan assures us that three outbreaks have occurred in eight weeks, each following the arrival of a boat or vessel. It has been suggested that the sudden contact of a people, under exceptional circumstances as regards food and occupation, with strangers, is the cause which seems to exercise an infectious influence on them, though what the nature of it is no one knows.

Again, on the river Amazon, speaking of the gradual exhaustion of certain tribes friendly to the whites who in-

habit the country near Ega, Mr. Bates says: "The principal cause of their decay in numbers seems to be a disease which always appears amongst them when a village is visited by people from the civilised settlements,—a slow fever accompanied by the symptoms of a common cold, *de fluxo*, as the Brazilians term it, ending in consumption. The disorder has been known to break out when the visitors were entirely free from it, the simple contact of civilised men in some mysterious way being sufficient to create it."

A still more recent, as well as a more strictly parallel, illustration of the occurrence of the malady in question in another part of the globe is contained in the account of the cruise of H.M.S. *Galatea* in 1867-68, where the following statement occurs:—"Tristan d'Acunha is a remarkably healthy island; but it is a singular fact that any vessel touching there from St. Helena invariably brings with it a disease resembling influenza. St. Kilda, off the west coast of Scotland, is known to be also similarly affected whenever a party lands amongst the people from any vessel. Whatever may be the real cause of the mysterious ailment—whether it is produced by contagion, like certain other epidemic diseases, or by a feverish excitement arising from a contact of a higher with a lower civilisation,—the actual occurrence of the distemper seems to be fully established; and the experiences of Ega and Tristan d'Acunha afford interesting illustrations of somewhat similar results in many different parts of the world."

Does not this seem to point to some subtle influence, the nature of which is unknown as yet? May not this in some measure account for the rapid decrease of the population of the South Sea Islands after contact with Europeans; and is it not pregnant with suggestive ideas as to the nature and the cause of transmission and diffusion of other epidemic diseases?

To return to cholera. In the present state of our knowledge we can only be guided by the inferences from well-ascertained facts and such laws as we have ascertained to be in constant operation, watching and carefully observing until we may, were it only by a process of exclusion, arrive at some deeper knowledge still. "So far," says the chief sanitary authority in India, "the history of cholera is full of enigmas and seeming contradictions, and though we have of late years collected many valuable data, and understood the importance of studying them on a broad basis, we know no more of the exact cause of the disease than our grandfathers did. We know that, whatever the cause may be, it flourishes

in the midst of insanitary conditions of dirt and overcrowding, and especially of impure water, impure from whatever cause; we know that it is liable to occur under certain conditions, and at certain times and seasons, and we should endeavour to extend that knowledge, and hope, as we do so, to arrive at the precise nature of the disease itself." But it may be well to remember that "expenditure of public money must take place only on observed facts and experience", not in accordance with theories. "It would be prejudicial to real sanitary work if opinions which have been promulgated in some parts of India, as to the cause of cholera being due solely to the state of the water-supply, were to take root."

It is most important that we should arrive at some definite conclusion as to the real nature of the disease, for it is impossible but that our conceptions on this subject must influence the sanitary measures that deal with it, and I believe the question must find its final solution in India, where the disease is always present in its endemic and seldom absent from the epidemic areas. The highest authorities—for both I have the greatest respect—differ *toto cœlo* on this subject. Is it that they are both right, though seemingly so opposed in their views? Like the knights who fought about the silver and golden sides of the shield, will they not change places and find why they differed?

I would venture to suggest that in India the inquiry might be pushed with more detail in regard to individual cases and outbreaks in certain limited areas; and that, on the other hand, one or more epidemiologists of European fame should be deputed to visit India and study cholera with the eminent men who have devoted so many years of close attention to it there. It might be, I believe it would be, that mutually they would gain from each other, and that those who went out would find their own views confirmed as to the disease in Europe—modified as to what they deemed it to be in India. Of one thing I am convinced, that simple truth is the object of their search; and I feel sure that from such combined action the greatest benefit would result.

I must now bring these remarks to a conclusion. I have made them purposely of a very general character, as I wished rather to indicate some of the subjects that we may profitably discuss than deal with them in a controversial manner. I should like to have spoken of other epidemics—fevers, small-pox, the exanthemata and other miasmatic or zymotic diseases; of diarrhœa, dysentery, influenza, diphtheria, which all interest us as epidemiologists, and of those peculiar indigenous and endemic diseases, such as leprosy, elephantiasis,

beri-beri, and a host of cachectic conditions generally attributed to malaria, but perhaps susceptible of other explanations; of the interesting discoveries of Lewis, Bancroft, Manson, and others which have revealed to us the existence of parasites infecting the blood and tissues of men and animals to an extent hitherto undreamt of, probably having important causal relations with some of the endemic diseases hitherto referred to other causes, and on which I have already had the honour of addressing you. But time does not permit me to do so, for I have already detained you too long, and I can merely refer to them as subjects that I hope may from time to time occupy the attention of the Society. It only remains for me to thank you for the patient attention with which you have listened to me for so long.

ENTERIC FEVER IN INDIA, WITH SOME OBSERVATIONS ON ITS PROBABLE ETIOLOGY IN THAT COUNTRY.

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[*Read: February 4th,* 1880.]

In venturing to bring the subject of enteric fever before this Society, I have to remark, at the outset, that my chief object in doing so is to offer, for your consideration, the results of my experience bearing upon the probable etiology of the disease, as it presented itself to me, in Europeans and natives, during my service in India. That it has long found a congenial home in our Eastern empire, may be gleaned from the classical works of Clark, Annesley, and Twining. But the distinguished honour of first identifying it there, on the perfected lines laid down by Gerhard, Stewart, and Jenner, belongs exclusively to Surgeon-Major Scriven, of the Bengal Medical Service, now civil surgeon of Lahore. Scriven had learnt the error of the prevailing opinions, which excluded the affection entirely from India and the tropics, from a *post mortem* examination, at Meerut, in November 1851, in which, the ulceration of Peyer's patches and other appearances characteristic of typhoid were unmistakably demonstrated, whilst the history of the previous symptoms of the case agreed fully with the view entertained regarding its true nature. (*Indian Annals of Medical Science,* No. viii, p. 512, 1857.) In 1854, he published some rough notes of three cases met with in Burmah. (*Medical Times and Gazette,* p. 70, January 28th, 1854.) We further learn that he subsequently saw, whilst serving at the European General Hospital of Calcutta, one or two doubtful cases which recovered, and three others, regarding the nature of which there never was any doubt, and in all these the diagnosis was satisfactorily confirmed by the revelations of the autopsies. (*Indian Annals,* No. viii, p. 521, *et seq.*) The subjects of the disease were young Europeans residing in such widely scattered localities as British Burmah, Lower Bengal, and the North-West Provinces.

In the latter part of the year 1855 I stumbled upon, and detected the presence of, enteric fever among the native prisoners in the Ajmeer jail, Rajputana. Three well marked cases, with an epitome of the symptoms, during life, and of the necroptic appearances at the inferior end of the ileum, were recorded and published in the seventh number of the *Indian Annals*. At the concluding part of that paper the following remark occurs: "I would further observe that, without the most scrutinising *post mortem* examinations being made in all those fatal cases which are denominated remittent fever, the conclusions drawn must be attended with some degree of uncertainty. Are not many of these examples of typhoid and typhus fever? When subsequently attached to the Megwar Bheel Corps, Kherwarrah, Rajputana (1857, 1859), I recorded several well marked cases among the Bheels of the regiment and the aborigines of the district. It prevailed in the regiment and surrounding villages, more especially in the autumn and early months of the cold or winter season, when the worst forms of malarious fever abounded. This experience, coupled with that obtained when I was employed as civil surgeon of Ajmeer, induced me to inquire, through the columns of the *Indian Lancet*, whether many of the malarious remittents met with in hot countries were not in reality either typhus, typhoid, or relapsing fevers, modified and often materially aggravated by the presence of malaria in the system. In 1860, during great scarcity, I noted the prevalence of relapsing fever; and that many of the so-called malarious remittents were simply typhoid or typhus fever modified, often for a time masked by malarious poisoning. I have repeatedly observed, ever since this declaration was made, both in and around Culcutta and elsewhere in India, in a very large number of remittents with diarrhœa and severe typhoid symptoms, the chief factor is the typhoid fever poison. When the blood is also surcharged with a large and effective dose of malaria, death often ensues from the combined operation of these causes before ulceration of the agminated glands occurs. This happened in three of my patients, residing in the suburbs of Calcutta in 1875, who succumbed in from eight to twelve days from what would formerly have been designated as remittent or jungle fever; but in all there was very marked prominence of the agminated glands. Had these patients survived long enough, ulceration would have been developed in due course. In both natives and Europeans, who have been subjected to the influence of malaria, the fluctuations of temperature in

enteric fever are greater than in those who have never been exposed to the action of this poison, the morning remission being so marked as to delude the observer into the belief that he is dealing with a malarious remittent simply when he is really called upon to encounter enteric fever modified by malarious infection of the system. In my practice at the College and General Hospitals, and among the various classes of the community at Calcutta, I have seen the disease in all its phases; so mild,—that persons have gone about their ordinary business during the greater part of the fever; so severe, as to end fatally in a few days, either singly, from an overpowering dose of the poison, or when complicated with serious malarious poisoning; or to terminate rapidly by uncontrollable hæmorrhage from ulcerated agminated glands, or by perforation of the gut and general peritonitis. In natives, the rash is not so easily made out as in Europeans; but, with care, it can often be distinguished in those whose shades of colour verge upon brown rather than black. In a certain proportion of Europeans the rash is not observed; but, even in fatal cases of this kind, the characteristic and unmistakable ulcerations of Peyer's patches are, nevertheless, found after death. Ever since Scriven directed attention to this subject, enteric fever has been recognised as a constant factor in the sickness and mortality at the General Hospital of Calcutta, to which he was attached for several years. The conduct of the disease has been most carefully watched, and fatal cases, whenever possible, tested by *post mortem* examinations. I extract the following information on this subject from my reports for 1873 and 1874.

	1865.	1866.	1867.	1868.	1869.	1870.	1871.	1872.	1873.	1874.
Cases of Typhoid	17	18	12	5	4	14	15	17	7	11
Died	3	3	4	3	3	1	5	11	3	5

In Scriven's discovery of enteric fever among young Europeans, and my recognition of it among natives, the first thing which attracted attention was the morbid anatomy of the agminated and solitary glands at the lower portion of the ileum. Its endemic occurrence has since been established and acknowledged by Edward Goodeve, Chevers, Fayrer, Morehead, Francis, Cornish, Furnell, Lyons, Moffatt, Massy, O'Brien, Cleghorn, Green, Hanbury, and a host of others, comprising many of the most able and zealous members of the Indian and British medical services practising in India. A place has been assigned to it in the statistical returns since 1871. The general result is that enteric fever now figures as the most fatal form of

pyrexial disease met with among the younger European soldiers sojourning in India. Year by year we have been hearing more and more regarding its extensive prevalence. So that, according to the statistics lately furnished by Dr. Bryden, there were, in the course of three years ending 1876, no fewer than 368 deaths among the European troops in India ascribed to the ravages of enteric fever. From the facility with which necropsies can be commanded in the British Army, most of these cases must have been verified by *post mortem* examination. Apart from the much greater number of recoveries which must have been observed, these fatal cases constitute a significant and important aggregate outcome of clinical work in the domain of diagnosis and pathology by a large number of medical officers, scattered over the length and breadth of the land, who were, before they migrated, in the ordinary course of duty, quite as competent from previous training, education, and experience gained in the medical schools of the United Kingdom, to diagnose enteric or typhoid fever, as to distinguish any other grave specific disease or fever included in our nosology.

The disturbing influence of malarious poisoning is not specially peculiar to enteric fever. During malarious seasons, it has to be taken into account in the diagnosis, prognosis, and management of many other morbid conditions, both medical and surgical. Every experienced physician and surgeon knows how materially convalescence or recovery from acute disease—local or general—is hindered or retarded by its predominating existence in the economy. This fact is, doubtless, equally well recognised by those having experience of the ailments occurring among persons who have finally taken up their abode in this country after protracted residence and service in the more malarious parts of tropical and tropoidal regions. But whilst acknowledging so much, there does not seem to be any valid ground for admitting that there is any more intimate relationship between malaria—the alleged potential cause of genuine paroxysmal fevers—and the poison or *materies morbi* of enteric fever, than there is between the former and the cause of any other affection whose course it may modify, complicate, aggravate, or even mask. In reviewing this department of the subject, it should not be forgotten that, in medicine, decentralization has been the tendency of our times. It behoves us also to bear in mind, that formerly it was the fashion to ascribe the invasion and development of small-pox, measles, and

scarlatina to the operation of a single causative agency; and that prior to the investigations of Bretonneau, Louis, Gerhard, Stewart, and Jenner, typhus, typhoid, and relapsing fevers were all viewed as springing from a common source. The result, however, of the accumulated observation, during the past thirty years, has been to demonstrate more and more clearly the non-identity of the poisons exciting typhus, typhoid, and relapsing fever, with as much precision and exactitude as clinical research had proved, long before, the non-identity of the poisons lighting up an attack of variola, measles, and scarlatina. Such being, with very general consent, the present state of scientific opinion, in this sphere of etiology, it is difficult to subscribe to the hypotheses recently promulgated that, on the one hand, enteric fever in India is simply one of the various expressions of malarial disease, and that it is only malarious, remittent, or continued fever with intestinal complication; or, on the other, that it may be the product either of a specific cause, or of malaria, or of both. The outcome of my own experience of enteric fever is that malaria—such as is the accepted cause of paroxysmal fevers—is never the cause of enteric fever in India. And though it is not, perhaps, necessary to accept in its entirety the pythogenic theory of its origin, or the spontaneous generation of the poison; yet, the intensely insanitary condition of the cities, towns, villages, and hamlets throughout India renders it inexpedient to dispense with the filth theory as unquestionably the best working hypothesis hitherto advanced to explain the endemic prevalence of the disease in that country; or to fall back upon the existence of two or more efficient sets of exciting causes for the same specific affection. There is, indeed, no more ground for allowing that enteric fever, as observed in tropical latitudes, can be roused into activity in the human subject by two or more sets of poisons, viz., marsh miasmata or malaria, and a specific poison, than there is for proclaiming the theory that any of the contagious exanthemata can be originated by more than one specific poison.

That of late years, or at least since 1862, the cause of enteric fever in temperate climates has usually been attributed to the existence of putrescent or decomposing filth of an animal nature is very patent from the published literature of the subject. The evidence thus available goes to show that, whether the potential poison is generated in such a nidus *de novo*, as was believed by the late lamented

Dr. Murchison, or not, it is, in its beginnings, in some way or other, intimately associated with the presence of animal matter or fæcal filth undergoing putrefactive metamorphosis. As it is probably with the cholera evacuations, so it may be with the excreta of enteric fever, the effective *materies morbi*, or germ, or poison—call it what we may—may not exist in a perfected and matured state, as maintained by the late Dr. Budd of Bristol and others, in these when they escape from the alimentary canal. But that, in both instances, the material exists out of which the effective poisons can be reproduced under favourable circumstances—it may be, as I have often thought, in conformity to the law of "the alternation of generation"—is quite within the region of possibility, if not, indeed, of probability. At any rate, the phenomena of this fever are constantly being reproduced, from the action of one single cause, in the same form, attended by a characteristic train of symptoms, and accompanied by a series of pathognomonic lesions, apparently very much alike in all parts of the world, in the white as in the black races, but subject, at certain seasons, to some modification, as has already been admitted in the foregoing observations, through the influence of malaria, temperature, and so forth, with as unerring certainty as is a potato or turnip, placed in a congenial soil, reproduced under the favouring condition of heat, moisture, and light, from their respective seeds, or as is syphilis or small-pox from their respective poisons or germs. On such a hypothesis as this, the generation of the disease and its dissemination to the predisposed through infected air, water, milk, and other articles of food—it may sometimes be by means of foul clothing, bedding, and the ill-ventilated and neglected rooms in which cases may have been treated—become intelligible.

When I detected enteric fever among the native prisoners of the Ajmeer jail, I attributed the outbreak to the extremely insanitary condition of the prison itself, and I had this corrected, as far as was possible with the means at my disposal. No word painting that ever was invented could exaggerate the abominable filthiness of the sleeping accommodation set apart for the convicts. They were arranged in groups of from fifteen to twenty, in narrow low-roofed cells, and packed far too closely together. A few earthenware vessels were provided as urinals and commodes. In this way, they were herded together from 8 P.M. to 6 A.M. daily. After the removal of the prisoners, generally, for out-door labour on the roads, the filth was taken away, but much of

it was washed into shallow channels, leading to a neglected cesspool, just outside the building, from which unspeakable odours emanated, on the south-western aspect; or, in such a situation, as to allow the effluvia issuing therefrom to be wafted back into the interior of the jail by the prevailing wind. The well from which the drinking-water was drawn was not far distant, only a few yards. In addition to direct sources of contamination, which were self-evident, it was so near the cells, drains, and cesspools, that it must have been exposed to pollution by percolation through the intervening gravelly soil.

In those days the pythogenic theory had not been propounded. Milk had not been regarded as a possible vehicle for the conveyance of the poison into the human economy. But, in relation to this latter point, I may here briefly narrate an incident that happened to me when civil surgeon of Ajmeer. A complaint having been submitted concerning the quality of the milk provided for the consumption of the prisoners, I found that it had a most offensive taste and smell. My native doctor suggested that this arose from its being mixed with impure water. We visited the village from which the milk was supplied. Some fresh drawn milk was then mixed with water obtained from a neighbouring stagnant pool, which was used for ablution and bathing by men, women, and children, and around which were the evidences of fæcal pollution. The mixture thus procured, corresponded, in its offensiveness of taste and smell, to that which had been objected to as unfit for use and condemned. On my urging these facts in the proper quarter the contractor was changed, and no more milk adulterated with stinking water was supplied during the remainder of my residence at Ajmeer.

Whilst at this station I met with cases of enteric fever among the population of the city. But confirmation of the diagnosis in fatal cases was not practicable owing to the prejudices of the natives. This unavoidable defect is all the more to be regretted because, as has already been pointed out, it too often happens that, in the very dark skin, the lenticular rose-coloured eruption, coming out in successive crops, and in all their phases disappearing on pressure, and quickly returning on the withdrawal of the same—such a valuable indication in the fair-skinned European—cannot always be distinguished. Here, again, there was no difficulty in discovering abundance of animal filth. In almost every house in the town there was a privy, and, in many, a cesspool in addition; the evil

odours emanating therefrom in close, still, and warm weather tainted the atmosphere within; whilst the drains in the streets receiving liquid sewage from overflowing privies and cesspools polluted it without.

When I was serving with the Meywar Bheel Corps, at Kherwarra, Rajputana, in 1858, the family of one of the leading native officers of the regiment, residing five miles from the station, was laid prostrate with what my native doctor considered to be remittent fever. Of five attacked, one boy and one girl died. The local and general indications, the duration of the disease, and the prolonged convalescence of those who recovered, conformed to the ordinary conduct of enteric fever. In a corner of a small courtyard adjoining the two rooms in which these cases were accommodated, there was a recess to which the females of the establishment chiefly resorted. Here was a privy reeking with sickening effluvia, and communicating with the exterior by means of a shallow blind channel. Here, as at Ajmeer, there was no lack of animal filth in a state of putrescence to explain the occurrence of the outbreak.

During my connection with the Medical College and General Hospitals at Calcutta, not a year passed without my seeing cases of enteric fever. It was most abundant in the autumn, then in the cold season, next in the hot weather, and least so during the monsoon. The modifying power of malaria was most apparent in the autumn and first half of the cold season, becoming less so in the middle and end of this, and scarcely perceptible in the remainder of the winter and during the hot months. Strangers of the dark and fair races below the age of thirty were most exposed to attack. It prevailed in the palaces of the rich and well-to-do and in the huts and hovels of the poorer classes. It spared neither sex. My impression, derived from unremitting attention to this subject, was that enteric fever was domesticated as an endemic in Calcutta.

Looking around for the cause of this, a little attention will show that the same insanitary conditions as are generally considered to lie at the foundation of the disease in temperate regions exist, to a very marked extent, in the metropolis of British India. Until within the last few years, filthy open drains lined the streets of the European quarter, as they did down to 1876, and do now in most parts of the native division of the city. Into these were drained the filth from kitchens, servants' rooms, stables, etc. In the lower rooms, close to these drains, the atmosphere was perceptibly tainted with effluvia. In most com-

pounds there was a servants' privy too frequently left in a neglected and insanitary condition. Prior to my departure in April 1876, some of these were connected with the sewers; but I could never satisfy myself that the trapping was sufficient to prevent back draughts; and when it is recollected how scanty—excepting during the monsoon—is the water-supply for flushing the sewers, the danger of such arrangements becomes readily intelligible.

In reviewing the sanitary condition of Calcutta, the material of which the sewer walls are constructed should not be excluded from consideration. The bricks are porous, and the interior of the sewer walls is not lined with any impervious cement to prevent oozing outwards or inwards. Thus, it is said, that it was part of the original design to make the Calcutta sewers drains as well as sewers, in order that they might help to carry off surplus water during the rainy season. That they do this, to a certain extent, by allowing percolation through their porous walls, must be admitted. This advantage, during the rainy season, from the 20th of June to the end of September, or sometimes to the middle of October, is reversed during the winter, and hot or dry months, from November to the 20th of June. During the long period of each year when the sewers are often imperfectly flushed, the percolation will often be from the sewer into the surrounding soil. Under such circumstances, deleterious exhalations must be elicited by the influence of a tropical sun operating with a continuity and brilliancy, during some of these months, which can only be fully realised and appreciated by those who have had substantial experience of the climate, polluting the air, and it may be the water-supply of those who, from caste prejudices, still decline to drink the filtered water of the corporation.

The sanitary defects of the native quarter are still more glaring. Both outside and inside the dwellings of the rich and poor; of the Rajah and his household; of the mercantile prince and millionaire and his humble dependents; an atmosphere charged with the products of decomposing animal filth is the rule. This portion of the city abounds with stagnant tanks, many of which are employed equally for bathing purposes, for yielding drinking-water, and as receptacles for all sorts and conditions of abominations and filth. In the suburbs, where dairies are maintained, there is too much reason to fear that such water is often used for the dilution and adulteration of the milk sold to the citizens. In the interior of most house compounds, or enclosures, private privies are kept up. During overflow, many of

these discharge themselves into the outside drains. But whilst these are being closed, as the sewers are extended, and the connection of the house drainage by no means keeps pace with this blocking-up of the drainage of houses, the effect must be very deleterious. These defects in the sanitation of native households are now receiving the vigilant attention of the sanitary department. Their eradication, implying, as it does, a complete revolution of the conservancy of these domiciles, is not even second in importance to the measures deemed needful for the improvement of streets, roads, and the provision of wholesome filtered water. In these cases, and they are not few, in which the filtered water of the city is not used, the wells and tanks are often situated in the immediate vicinity of latrines or privies, and are thus liable to become polluted, either directly, or indirectly, by percolation.

The reports of the sanitary commissioners of the various provinces, from Rangoon to Bombay, and from Peshawur to Cape Comorin, are constantly referring to the existence of the deplorable sanitary defects I have thus ventured to pourtray from my own personal observation.

But we are met with the assertion that such insanitary conditions have no existence in military stations, barracks, and hospitals. Before accepting this view, without some reservation, accurate and continuous information is desirable regarding the condition of the privies used by the natives in the service of officers, and of the latrines employed by the numerous followers of native and British regiments. Even if the sanitary excellency of these is unimpeachable, there is still overwhelming evidence, in the reports of the Sanitary Commissioners, to prove that the neighbouring bazaars, frequented more especially by the younger soldiers, present, in unmitigated intensity, all the insanitary blemishes summarised in the foregoing narrative. It is here, it may be far away from their barracks, where they are not only liable to breathe air impregnated with emanations from decomposing animal filth, but obtain food, milk, drink, and water, of questionable quality and purity.

Whilst, therefore, it may be tolerably correct to declare that, as a rule, the source of typhoid or enteric fever in India may not be traceable to the barracks occupied by European troops, on account of their scrupulous cleanliness, there is still sufficient reason, when all the circumstances of the case are duly balanced and considered, for provisionally attributing the wide spread occurrence of enteric fever in India to the same source or causes which, in some

way or other—not, perhaps, as yet, quite clearly understood—are considered potential in its production in this and other temperate climates. If animal filth, or fæcal filth undergoing putrefactive decomposition does not exist to any great or appreciable extent in barracks and hospitals, and their immediate vicinity, it does nevertheless abound, in marvellous profusion, in bazaars, villages and hamlets, freely visited by the soldiery. There is nothing, therefore, connected with the prevalence of enteric fever in India, very repugnant to the Murchisonian doctrine that, in some way or other, in its etiology, it is closely associated with the presence of putrefying animal or fæcal filth. The reason why this form of fever is not more frequently met with, amid such an abundance of fæcal filth in a state of putrescent fermentation, is probably to be discovered in the fact, that, fortunately, most of it is not pent up in ill-ventilated sewers, but exposed daily to the powerful antiseptic influences of the atmosphere and sunlight in open drains, open privies and latrines, and comparatively open cesspools. The impression which has been gaining ground, of late years, that, as the sewering of Calcutta has been advanced, so has enteric fever been more frequently observed, lends some countenance in support of this view. This augmented prevalence, however, may, in some measure, be owing simply to greater recognition of the fever, although it must not be forgotten that attention has been directed to the subject during the past twenty or thirty years.

One of the arguments used to support the view that enteric fever, among the British troops, in the Madras command, is only remittent or continued fever with bowel complications, is the difficulty which has apparently been experienced in tracing the origin of the cases to the existence of pythogenic causes. In this matter, if due care be taken, there need be no supreme obstacle, seeing, as has been pointed out, how very common such filth conditions really are in the Indian peninsula. But the difficulty, especially in the case of those who dwell in clean and well cared for barracks, is much magnified by the considerable period of incubation or latency of the poison in the system. According to the best authorities, this varies from seven or ten to fourteen or twenty days in most cases; so that to trace the origin of any case, the investigation must be of a much more searching and comprehensive character than any which has hitherto been conducted in India. It is manifest that the generally acknowledged freedom of the

barracks and their immediate surroundings from putrescent fæcal filth is, in itself, scarcely sufficient. The inquiry should embrace a calm and impartial review of the patient's history and habits, derived, when possible, firstly, from himself, and, secondly, his friends, and extend over a period of many days antecedent to the attack. As the disease is rarely spread by direct contagion, or from the sick directly to the healthy, in the well ventilated military hospitals in India, it follows that, in almost every series of cases, a similar exhaustive investigation would have to be undertaken, if the source of it, in all cases, is to be identified to the utmost extent attainable.

It is curious to note the nature and character of the opposition to the views of those who contend for the endemic and widely scattered existence of enteric fever in India, inasmuch as it, in some measure, resembles that which Stewart and Jenner encountered when they succeeded in splitting up common continued fever into three distinct and well-defined diseases. Then, a reference to the literature of the period shows that enteric fever was regarded by many eminent physicians as common continued fever with bowel complication, just as the enteric fever in the Madras command has, of late, been considered by Surgeon-General Gordon to be merely "malarial", or climatorial fever of the continued type, with intestinal complication. As it was in the former case, so it will probably be in the latter, the antagonism will do no harm. It is more likely to do good, in stimulating clinical research, bearing upon the diagnosis and the etiology of the fever. Should the ultimate results of further inquiry, thus promoted, go against the pythogenic theory, such a conclusion, however well founded, would not suffice to justify us in disputing the domesticated existence of the fever in India. It would have the effect of causing investigation in some other line or lines, with a view to localise or to discover the habitat and nature of the 'cause not only in that country, but in Europe and America. But this hypothetical position would, in no way, alter our notions regarding the nature, symptoms, pathology, and morbid anatomy of the fever, provided, as no doubt they would, its clinical phenomena and morbid anatomy remained as well-defined as they are, in all parts of the world where it has been identified at the present time. Doubtless, the genesis, the comportment, character, and habitat or habitats of the principle or poison developing, under auspicious conditions, in the human subject, and a few of the higher mammalia, the pathological phenomena

of enteric fever, are matters of supreme importance to the sanitarian and state physician. They also concern the clinical physician in the domain of prophylaxis, hygiene, and in the management of any given case; but to him in the field of practice, the product of this principle, poison, or cause, viz., the *dothenenterite* of Bretonneau, the *febre typhoide* of Louis, the typhoid fever of Stewart and Jenner, the pythogenic fever of Murchison, and the typhoid or enteric fever of our present nosology, is the thing itself in the concrete upon which his diagnostic, prognostic, and therapeutical skill must always be concentrated. Whatever views may be entertained concerning its etiology, so long as the thing—enteric fever—is present in India, it will, now that so much attention has been attracted to it, continue to be more and more generally recognised as a distinct entity (generated and diffused, as I believe, independently of malaria, though like many other affections, being liable to be modified by it), perfectly irrespective of the views that may, from time to time, prevail about its precise etiology.

Should the eventual result be, as I believe it will, the still further amplification of enteric fever cases, at the expense of the group of remittent and continued fevers, this expression of progress in differentiating it, in noting its pathology and morbid anatomy, is, in no sense or degree, calculated to impugn the excellency, ability, and devotion of those who are slow to admit the advancement thus claimed in this department of medical science. As well might the immediate predecessors and cotemporaries of Ambrose Parè, Harvey, Jenner, Laennec, and Simpson feel disparaged because to them was not assigned the task and duty of discovering the uses of the ligature, the circulation of the blood, vaccination, the stethoscope, and escape from pain by means of chloroform, ether, and laughing gas. As it was with reference to these discoveries, so will it be with regard to that of enteric fever in India. Both the older and the younger medical officers have been, and are, labouring for the common good. The result of these labours has been a great accumulation of facts, and consequent extension of knowledge, followed by an improved application of the same to the clinical study and diagnosis of enteric fever, no less than to measures to be adopted for its eradication or prevention.

Before drawing these observations to a close, I would beg to enter a protest against the mode now adopted in jails and military hospitals in India, of disposing of the excreta derived from cases of enteric fever and cholera. Deodorized by dry earth, these are buried in trenches, often

close to the habitations of the healthy: and the soil thus richly fertilized, is utilized for the cultivation of vegetables and cereals. This method is, no doubt, sufficient for the destruction of ordinary night soil. But it is questionable whether it affords adequate protection against local outbreaks of enteric fever and cholera. By this plan, deodorization is wonderfully complete. Still, if there be a solid basis of truth in the theory that the poisons or things causing cholera and enteric fever, respectively, are contained, in some form or other, in the dejecta cast off in the course of these diseases, and that the soil, under certain conditions, favouring the stagnation and impurity of its contained moisture may, according to Pettenköffer and his followers, constitute a congenial nidus for their subsistence, development, and propagation, subsequently, through the medium of air, well water, milk, and articles of food, there would be reason to fear that the dry earth, admirable as a mere deodoriser, may not be a disinfectant in the sense of destroying, or rendering inoperative the potential causes of these maladies. Carbolic acid, chloride of zinc, and sulphate of iron solutions, are probably efficient if employed in sufficient quantity, for the accomplishment of this purpose. On a reference being made to me some years ago, on this subject, by the Lieutenant Governor of Bengal, I advised that, in the absence of these disinfectants, the prompt incineration of the alvine discharges in cases of enteric fever and cholera, would prove a safe and effective prophylactic measure.

Whilst, in conclusion, I have to thank this meeting for the patient manner in which the foregoing observations have been received, I must not omit to tender my best acknowledgments to Sir Joseph Fayrer, K.C.S.I., and Surgeon-Major A. Clarke for the opportunity they have kindly afforded me of referring to several recent and able Reports bearing, among other things, upon the Etiology of Enteric Fever in India.

ON THE ENDEMIC CONTINUED FEVERS OF SUB-TROPICAL LATITUDES.

By Surgeon-Major W. G. DON, M.D.,
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[*Read: March 3rd*, 1880.]

When I had the honour to be asked to appear before you to-night I hesitated, for I doubted my ability to bring before you in any new shape a subject sufficiently cognate to the work of this learned Society. I am far from hinting that my presence here necessarily shows I satisfactorily resolved these misgivings; rather I venture to mention them at once as a plea for shortcomings, and for indulgence at your hands. The title of my paper of course broadly indicates its scope: but, as that opens up a wide field of doubt and controversy, it is necessary for me, as a preliminary matter, to explain what are the special points upon which I wish to engage your attention.

The first question obviously is, what do I mean by the endemic continued fevers of subtropical latitudes? Now, I am not about to essay the impossible task, in a short paper, of attempting any widely comprehensive survey or scientific classification of these fevers. I can only touch upon their more salient points. I intend simply to follow the arrangement and nomenclature of the *Army Medical Blue Book*. I am aware that exception may be taken on various scientific grounds to the grouping together in the *Blue Book* of various forms of fever under the heading "continued"; but I waive the discussion of that point, and follow these army returns wholly and solely because they afford us data nowhere else to be found.

The Continued group of fevers in the *Blue Book* may be taken to include only—

a. Febricula;

b. Simple continued fever;

c. Enteric or typhoid fever, as it is synonymously called.

True typhus, did it occur, would be included, but fortunately we now know little or nothing of it in the army—at all events, in the sub-tropics. Yellow fever, on account of its

special importance, finds a heading in the *Blue Book* for itself, and, therefore, will not concern us except incidentally. The exanthemata proper are wholly excluded from my remarks; so also the paroxysmal, or purely malarial fevers, which, however, are often inextricably mixed up with the continued forms, and even, as I imagine, produce hybrid types with them.

To a consideration of the three first named forms of continued fever, therefore, I shall limit myself, while I endeavour to expound what appears to me to be their mutual relationship and etiological affinities.

The second question naturally is, what do I mean by sub-tropical? The term sub-tropics is more a geographical expression than anything else. It is applied to those belts of the earth's surface which, while neither strictly tropical nor temperate, are yet both; that is, tropical in summer and temperate in winter. This region lies, broadly speaking, between the 20th and 40th parallels of latitude north and south of the Equator. But it cannot be bounded by rigid geographical lines; it is much more correctly indicated by the isothermal, or still more closely by the isotheral markings, which show the areas of like summer temperature. It is temperature that determines the true sub-tropics of either animal or vegetable life, and of disease. Especially is this seen in the endemic fevers, for summer heat is undoubtedly the chief factor in their genesis and evolution. For this reason, the general question of sub-tropical meteorology is of great importance to us as epidemiologists; for I believe that, to broad climatic causes, rather than to mere local insanitary conditions, must we look for an explanation of much in the etiology of the endemic fevers.

As yet we are comparatively ignorant as to how the imponderables—heat, light, and electricity; the variations of atmospheric pressure, or the rapidity or amount of evaporation and radiation affect vital processes in the human body; but that such forces exercise most important influences is a self-evident fact. The subject, however, is too recondite for discussion here; I merely mention it in order that we may not overlook it. We have in this Society lately heard a good deal about certain forms of endemic fever in India, and, in order that the same ground may not again be gone over, I shall almost avoid reference to that country.

I propose to illustrate my subject by referring chiefly to two of our army stations, with which I happen to have a personal acquaintance, and which afford excellent examples

of the subtropical fevers under consideration. I mean Bermuda and Gibraltar, of which the following is a brief sketch. Both of these places lie between 30 and 40 deg. north, and are, therefore, well within the subtropical belt. Longitudinally, they are separated by nearly the entire breadth of the Atlantic Ocean. Surrounded by a warm sea, Bermuda is strictly insular; while Gibraltar, with great arid tracts of land north and south of it, is only partly insular. Although, therefore, the seasonal periods of the two places are nearly identical, their climate is by no means alike. The chief difference is, that Bermuda is humid and rainy all the year round, while Gibraltar is rainy only in winter and spring, and very hot and parched during the summer months. The summer temperature of both ranges between 78 and 90 deg. F., but there is considerably more diurnal range in Gibraltar than Bermuda. I have known the temperature in the latter place only vary a few degrees between day and night for months; all the time remaining at the steady high reading of 84 deg. or thereabout. In winter, Gibraltar is often 10 or 15 deg. colder than Bermuda; but frosts are unknown in both, except sometimes at the very top of the rock of Gibraltar. The general climate of both places is disagreeable and trying in summer, but most pleasant and very fairly healthy in winter and spring. The physical and geological structure of these two places is very unlike.

Bermuda—or, rather, *the* Bermudes—are a cluster of coralline islets, perched on the summit of the loftiest submarine mountain system we know of, for the greatest depths of the Atlantic lie quite close to them. They have a scanty soil, covering the loose coralline rock, which is usually so soft that it can be cut with a hand saw, and so porous that water soaks through it like a filter. Its surface, however, by the action of rain and air, soon becomes coated with a hard impervious stalagmatic crust. There are no elevations over two hundred feet, and no springs; wells sunk to the sea level yield only brackish water by percolation. The palatable drinking water is, consequently, from cemented tanks cut in the rock, and filled by rain catches, chiefly the roofs of the houses. Earth-closets and water-closets exist in the military and naval barracks, and in a few of the private houses; but the latrine is generally a mere cesspit in the porous rock, which allows the fluid portion of the excreta to filter away, leaving the more solid in the form of a pasty mass.

The white population are a vigorous race; many of the

families boasting an untainted English descent since the original plantations of 1632. The dark race are of all shades of colour, but are by no means a poverty-stricken people, but fairly well off, and very independent.

Gibraltar, on the other hand, is a huge solid mass of limestone, towering up some 1,400 feet, and having only a small habitable margin at its western base and southern extremity. The bare rock is full of fissures filled with vegetable detritus, and yields no springs proper. As in Bermuda the drinking water is from rain tanks, although the very brackish water obtained from an artesian well in the sandy isthmus connecting it with Spain is also now drank. The general sanitation is very carefully attended to, and quarantine often most rigorously enforced. Of late years a complete system of main drainage has been in operation; the drains are flushed by water pumped up into reservoirs and ventilated by shafts carried up the face of the rock above and beyond habitations. When this drainage system was inaugurated, many hoped the notorious fevers of the rock would disappear. I am sorry to say such expectations have been by no means realised. I mention this fact with no wish whatever to discredit such sanitary effort, but to show that mere local insanitation does not seem to be the chief factor in subtropic endemic fever.

The population of Gibraltar—over and above the garrison of five or six thousand men—is a very mixed one, and in some respects semi-oriental; but it is under thorough police supervision, and overcrowding and insanitary nuisance of any sort strictly guarded against.

I have sketched these two places just to show that in a physical or sanitary sense they have very little in common. Yet, when we come to examine the general form and type of the endemic fevers common to both, we find they are essentially the same.

This observation and contrast equally applies to Malta, Hong Kong, Mauritius, the Cape, and many parts of India. In my opinion the continued forms of fever in all semi-tropical countries are really almost identical; what varies their type and symptoms is malaria; according to the amount and potency of that element so do the continued fevers assume a remittent, or even relapsing, or other anomalous form. I now come to review, very shortly, the three forms of fever comprised in the Continued group in the *Blue Book*.

1. *Febricula.*

This form has a variety of names, of which are—*a.* Ephe-

meral fever, from its shortness; *b.* Ardent (especially in India), from its sharpness; and, *c.* Broken-bone, in Bermuda, the West Indies, and Southern States, from the severe neuralgic symptoms which very often accompany it. Whatever its name, the essential feature of febricula is, that it lasts so many hours, or days, and is neither fatal nor complicated. It is *the* climatorial fever which sooner or later almost certainly attacks all Europeans during tropical residence.

Its commonness and occurrence under every variety of circumstance has induced writers to ascribe it to almost anything or everything of the nature of an exciting cause. My impression is, that it may be held to result, in a general way, from a temperature too elevated for the constitution and habits of the European, whereby, the balance of the eliminative functions, especially between the skin and the kidneys, becomes upset. My opinion further is, that if we could only explain the true pathology of this simple fever, we would get an initiatory and direct clue to the pathology and etiology of the more severe and dangerous endemic continued forms. Besides its constant and frequent occurrence in hot weather it sometimes prevails epidemically, and then as often attacks the natives—white or coloured—of the semi-tropics as it does European immigrants.

This has been well traced by Inspector-General Lawson in his researches on pandemic influences; to give one example. In the West Indian yellow fever area broken bone febricula has been observed to preceed that terrible disease: thus, in Bermuda the continued group of fevers (of which febricula would form 80 or 90 per cent.) gave in a series of years the average figures of 55·3 in an annual ratio of 1000 of mean strength; but in 1863, which preceeded the yellow fever epidemic of 1864, the ratio rose to 153·6, or nearly three times the average rate.

The epidemic prevalence of slight fever is therefore always viewed with apprehension in Bermuda, as probably a prelude to an outbreak of the much dreaded yellow fever. Febricula usually is resolved in a few days, but I have seen marked exceptions to this in the sub-tropics, where imperfect defervescence slowly drifted into a more continued or even an enteric form.

It is essentially a summer fever; no doubt cases are sometimes seen in the cool winter season, but such are usually directly traceable to alimentary indiscretions or downright inebriety.

2. *Simple continued fever.*

When febrile symptoms do not resolve within a few days, but go on and on for several weeks, and yet without any specific complication, the disease is called simple continued fever.

Some medical authorities maintain that the term "simple" is not only a misnomer but a paradox. For, say they, the very fact of continued febrile symptoms indicates a lesion somewhere; and therefore the term simple—meaning uncomplicated—is inapplicable. I will not argue this abstract point, but merely remark from my own experience, which is that of hundreds of others, that in semitropical countries, cases of endemic continued fever occur in which, throughout a protracted illness, the most careful diagnosis fails to detect any constant visceral complication.

Cases of this sort are often seen in Bermuda, where there is no malaria to mask symptoms. But in such places as Gibraltar or Malta, where malaria exists, the continued fevers are generally more or less complicated with it, producing a hybrid form of disease which has received a variety of names. Marston calls it gastric-remittent.

I am speaking now of cases without enteric complication. The simple continued fever on which malaria is engrafted is a most intractable although not a fatal disease; I have seen it distinctly relapse; it is generally followed by persistent rheumatism and neuralgia, and what has been curiously named "pernicious" anæmia; and such sequelæ are a source of much inefficiency and invaliding in our Mediterranean garrisons.

3. *Enteric or Typhoid Fever.*

This is the last form of fever under review in the continued group of the *Blue Book*. It has a special importance in being the fatal form, but has, besides, a very special interest to epidemiologists because of the controversy which has centred and is still raging round it. At our last meeting Surgeon-General Ewart ably introduced a discussion on this disease as it appears in India; and we also know the very able and exhaustive reports of Surgeon-General C. A. Gordon, on the disease within the Madras command. I know of no disease which has been more strongly dogmatised upon than enteric fever; a few years ago it was perfect heresy to question, however mildly, the views of eminent men who were the great exponents of the disease in England.

Now, however, doubts are pretty freely expressed on the causation of the disease, especially by army medical officers, whose experience of it extends to all parts of the world. Buckle, in his *History of Civilisation*, says: that, as unques-

tioned authority begets stagnation and deadness, so honest doubt is the parent of all progress and reform. Unquestionably the doubters on this subject of enteric fever are honest, and it is quite certain they have their wits about them.

The great thing for disputants on abstruse scientific subjects, who debate not as mere partisans, but with the "open mind", is first to understand each other. Let us therefore come to some understanding as to what we severally mean by the term enteric fever; otherwise, if we begin by mutual misapprehension, we are likely to end in a mere dispute over words and names. Let us proceed thus: forgetting theory and even setting aside authority, let us agree to a mere anatomical or pathological definition of enteric fever—that it is a continued form of fever presenting certain signs and symptoms, of which the chief and essential one is *infiltration, inflammation, and ulceration of the solitary or aggregate glands of Peyer in the intestines.* This definition merely recites pathological facts and commits to no theory of causation, specific or otherwise; anyone might subscribe to it. The fever, of which the above is the characteristic complication, has of late years figured very largely in military statistics as a source of much mortality in all our semi-tropical stations. But there was a time—and that very recent—when the disease now named scarcely figured at all in these statistics.

Are we therefore to conclude that it has only become endemic in all our stations from east to west during the last quarter of a century? The medical history of these stations does not countenance any such supposition. It is now returned as one of the chief causes of mortality in our intertropical military stations. But was there no mortality from fever there before the term enteric came into use? Of course there was, and at times very grievous; but men died under continued merely, or remittent. The conclusion is therefore irresistible, that the chief change in the fatal type of endemic continued fever is *only one of name.*

This change of name undoubtedly indicates an advance in pathological knowledge, but how far it necessitates an entire etiological change of view is another matter.

I need not trouble you with a recital of the well known pathognomonic signs and symptoms of enteric fever. Suffice to say, that in the summer of 1878 I saw about a dozen cases in Gibraltar, all of which presented the characteristic symptomatology of the disease, just as we might see it in London, and examination of the fatal cases revealed the essential bowel lesions. My experience in

Bermuda, however, is considerably different. During a three years' tour of service there, in charge of the Royal Engineers, I saw many enteric fever cases both in my own corps and in the regiments quartered in the island. But in a large proportion of these cases the symptoms were not altogether pathognomonic; for instance, I cannot honestly say I ever saw the characteristic rash; nor were the stools ochry, but nearly always green, dark or bilious. Some may say these were not cases of true enteric fever; but I answer, if not, what were they? For in the fatal cases the essential bowel lesion was found; nay, some even died of perforation.

I have thus rapidly glanced at the three forms of fever constituting the Continued group; let me now, as shortly, review some points in which they seem mutually allied and intermixed, both in their etiology and epidemiology.

The *first* broad fact in their mutual relationship is, that the early symptoms of these fevers are usually so much alike, that it is often quite impossible on admission to hospital to determine what form may ultimately supervene. Many times have I seen cases admitted under febricula, then changed to simple continued, and finally to enteric, just as the fever developed. This arose from no slovenly diagnosis in the first instance, but was forced on by the supervention of phenomena which it was quite impossible to anticipate.

The *second* broad fact is, that they occur simultaneously, concurrently and mixed up, at the same time and place, in the same regiment or community. It is a plain fact that, from the same barrack, or company room, one man may be admitted into hospital with febricula, a second with simple continued, and a third with enteric, all on the same day.

A *third* very important broad fact is, that all these fevers have the same seasonal period of prevalence. During winter and spring they are at a very minimum, or wholly absent; from June to October is the season of their prevalence north of the line, with a corresponding reverse period in the southern hemisphere.

Now, when such allied forms of disease as these fevers present such points in common as place, circumstance and season of occurrence, may we not reasonably infer that their causation must have also much in common? If the exciting causes of these fevers are quite distinct, who can differentiate them? If the subjection of men to apparently identical influences, results in the evolution of three different forms of fever, what explanation have we to offer? I confess such questions have often puzzled me exceedingly.

I have met medical officers who allowed that the etiology of febricula and simple continued was probably identical, but that enteric in its causation seemed to have *plus something more*. Whether this something more was essentially specific, or might be evolved *de novo* from pythogenic sources, they never would say. My own impression is, that, with varying degrees of intensity, the exciting causes of all these forms of fever, are, if not identical, entirely similar. I also believe that the chief factors of these endemic fevers are to us intangible, and purely climatorial. Local insanitation greatly aggravates and intensifies the type of the fevers, but it is not the main originating cause. I may be asked, If the general causation of these fevers is so vague, indefinite and non-specific, on what principle can their different forms be explained? In other words, why should one man get one form of fever, a second another, and a third the worst of all? Now, I doubt if such questions can be answered by reference to supposed exciting or external causes; I think they can be better explained by a study of internal or predisposing causes. Of predisposing causes I reckon three:

1. Constitutional, hereditary or acquired.
2. Age.
3. Want of acclimatisation.

Let me touch on these. The successful practical study of disease involves: first, the diagnosis of it; and second, the individualising of the subject of it. In endeavouring to carry out this principle with regard to these endemic fevers —particularly in Bermuda—I made several generalisations. I thought I noticed, that, while all and sundry were liable to febricula or simple continued, only those who presented the physical evidences of constitutional struma or tubercle were the subjects of enteric. The robust, big chested and vascular man, often a hard drinker, did get fever, but not enteric. The man of altogether reverse physical type and development, often temperate, or even teetotal, was the victim of enteric in an especial degree. If such generalisations were real and not imaginary, could it be that exciting causes, operating through inherent predisposition or tendency, would in the one case set up a simple uncomplicated form of fever, and in the other incite to a most dangerous form with glandular bowel lesion? It has been observed, even in this country, that some families seem to have a peculiar liability to enteric fever; if such is the case, it cannot be accidental, but might fairly be ascribed to constitutional proclivity of the kind I have mentioned.

That age, that is, youth and immaturity, strongly predisposes to all forms of endemic tropical fever, is a fact long and well known. That enteric fever, even in this country, is mainly a disease of youth, is also well recognised. What may be the true explanation or *rationale* of this fact it is more difficult to determine. I imagine it to be on some principle of natural selection or weeding out. It is worth noting that the age at which there is the greatest liability to enteric fever closely corresponds to the period of life in which there is the greatest proclivity to the development of struma, tubercle, and such like diseases of low nutrition. Intimately, and, as far as soldiers are concerned, inseparably bound up with age proclivity, is the predisposition to tropical endemic fever seen in the unacclimated. Now, the young soldier is necessarily the most recent arrival, and the most recent arrival in a tropical station is almost invariably a young soldier. So that in military statistics, age, and period of residence, are usually inseparably blended. This is well shown in the following statistics (not yet published) for British troops in Bengal for 1878, for which I am indebted to Deputy Surgeon-General Tydd. There were 166 deaths from enteric fever, reported from nearly every division, and, indeed, from nearly every station, in the Bengal Presidency during 1878. This was nearly three times in excess of the average of previous years. Three-fourths of the deaths took place in the second and third quarters of the year; that is, say between July and December. Nearly 50 per cent. of the deaths took place among men during their first year in the country, and nearly 60 per cent. among those under twenty-four years of age.

From these few facts and figures it is very evident that the process of natural selection, or weeding out of the most susceptible subjects to endemic causes of disease, is a rapid one. Seasoned troops, in short, are just those well weeded by death and invaliding.

Time does not allow me to enlarge further on this part of my subject, and I therefore propose to finish this paper by offering a few remarks especially on subtropical enteric fever.

I begin by assuming that we are already tacitly agreed that, in any case of continued fever in which the specific anatomical bowel lesion is recognised, we are justified in applying the term enteric to that case whatever may be its assumed cause. Starting from this hypothesis, I cannot help thinking that the said enteric lesion is to be found in several forms of a closely allied fever, and, at the same time,

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this bowel lesion does not necessarily imply a specific causation under every circumstances and in all parts of the world. It is only on some such assumption that we reconcile the mass of conflicting testimony and experiences which have of late years accumulated from all parts of the empire. Might we not reason that enteric fever occurs both epidemically and endemically? We have the support of analogy in this inference; for yellow fever, cholera, and plague, may appear either epidemically or endemically within certain areas. We know the theories of Budd and Murchison as regards this disease in this country; and I think we must conclude, with them, that in whatever mode the disease originates, when once set going, it tends to multiply and propagate itself in directions suitable for its spread.

To ignore the many proved instances of direct propagation, would, I think with Sir Thomas Watson, be not only "erroneous but dangerous". The disease, therefore, usually shows a distinct epidemic tendency in this country, and in temperate regions generally. But one may willingly subscribe to the main doctrines of the eminent exponents of enteric fever in England or elsewhere, without, at the same time, ignoring or shutting our eyes to our own experiences, and those of many others in tropical countries. No one I suppose denies that enteric fever may be introduced into a tropical country just as into a cold one, and there spread by propagation; indeed, in these days of rapid inter-communication, this is very likely. Nor will it be contended that, if its origin is pythogenic, it may not appear in any country where atmospheric conditions are favourable. But the question is, when there are no evidences of either direct or indirect propagation, and no ground for the assumption of a filth origin, *de novo*, how are we to account for the persistent and periodic evolution of the disease in a sporadic form in many semitropical areas?

Propagationists may say the specific germs are always and everywhere about, and ready to develop under favourable conditions of temperature, etc., and that the fault of their non-detection rests with the observer; that the links of a direct chain of causation can always be traced if sufficient pains and science are brought to bear on the investigation. The outbreaks of typhus in crowded armies in former days have been explained by the same theory, namely, the ubiquity of specific germs. But I hold that the theory of the ubiquity (and indestructibility) of these inferred germs is based on pure assumption.

Can it be logically maintained that these germs exist not

only in every station in India, and in such old places as Malta, or Bermuda, or Gibraltar; but even confront us when we penetrate into the virgin wilds of Zululand? For, in the late Zulu war, cases of enteric fever occurred in detachments of our young soldiers which no theory of specific contagion could explain, and which were usually vaguely accounted for on the pythogenic theory of foul drinking water. In short, I think there can be no doubt that enteric fever cases constantly crop up in hot climates, the causation of which is inexplicable, either by a theory of propagation, or of pure pythogenesis.

In such a statement I think I am but expressing the views of Surgeon-General Gordon and many officers of similar wide experience in the army and Indian service. If I mistake their views, at all events I am trying to state my own. In support of these views let me mention one or two instances which have come under my own observation. The 15th Regiment landed at Bermuda, in a healthy condition, from New Brunswick, just at the beginning of the hot weather of 1868. It was, of course, an utter mistake to land them on a tropical island at the beginning of summer, and in six weeks time this fact began to be painfully apparent. They were split up in detachments here and there on wild spots in Bermuda, making a military road. First diarrhœa and mild dysentery showed itself, and, almost simultaneously, febricula, simple continued, and many cases of enteric fever occurred. Meanwhile, the other troops in the island, who were more or less acclimated, had only the usual and moderate amount of summer endemic fever amongst them.

The detachment of the 15th which suffered most—especially from enteric—was encamped on perfectly virgin sand, on the sea shore, far removed from habitation, or even the very suspicion of fæcal contamination. As regards food and water, although not shelter, they were on a par with other troops who did not suffer from enteric fever except in the usual sporadic way. In fact, there was nothing to account for the fever, except the circumstance that the men were brought to Bermuda at the wrong season of the year, and perhaps imprudently placed under canvas; in short, the cause was nothing special, but a general climatorial one.

The principal medical officer ascribed the sickness of the regiment to "the effect of a sudden change from the cold bracing climate of New Brunswick, to the hot relaxing climate of Bermuda." The surgeon of the regiment,

referring to the prevalence of enteric fever in another detachment, said he "found it impossible to lay his finger on any specific cause for the prevalence of this fever". I can well endorse this statement, for I was in Bermuda at the time, and know how careful the possible and probable causes of the fever were looked into.

I will next mention an individual case in Gibraltar which came directly under my own observation, and which is chiefly remarkable that it occurred in a soldier who had actually been under lock and key for several weeks before he was attacked with enteric.

A young gunner of the Royal Artillery, of fair physique, but of rather delicate strumous appearance, was made a prisoner for insubordination, and placed in the guard-room, where he remained about a week before he was tried by court-martial. His sentence consigned him to the military prison, where he had not been above six days when he was discovered to have a large inflamed ulcer of the right leg. For this he was sent to the prisoners' ward in the station hospital and came under my treatment. In this ward he was kept under lock and key, with a sentry on the door; there were also one or two other prisoners with slight surgical ailments in the ward. From the condition of his leg it was necessary to keep him in the recumbent position, so that he was in every way under strict supervision. His diet and drink in the meantime was part of that in use by everyone in the hospital. At the end of three weeks the ulcer was healed and I was about to discharge him back to prison. He was, however, suddenly seized with acute febrile symptoms, like a smart attack of febricula. In a few days all the signs and symptoms of a severe attack of enteric fever developed. This man had several distinct relapses, and at the end of three months was little more than convalescent; he, in fact, just escaped with his life. Now the points of this case were, if this man was specifically infected before he was made a prisoner, the period of incubation of the disease was five weeks; if in the guard-room, four; and if in the prison, three weeks; if he caught it in the hospital it was in spite of his being under the very strictest surveillance, and wholly removed from any conceivable source of contagion either within or without the hospital.

Over and over again, in Bermuda and Gibraltar, I have seen isolated cases of enteric fever, both in barracks and private houses, which a filth theory could not logically explain. For if such cases arose from soil or water pollution, how came it that scores of other people living

under identically the same conditions were not similarly affected? Or, if soil and water contamination is the cause of the disease, why should it be so only in summer and autumn? Such filth sources of fever exist equally all the year round; as much in the mild temperate winter and spring as in the hot summer. If a cesspit system, and a porous absorbent rock, be at the bottom of the mischief in Bermuda, such cannot be in Gibraltar, where there is impervious limestone and elaborate main drainage and sewage conservancy. And so, by a process of logical involution, the etiology of enteric fever in the tropics becomes a subject of exceeding difficulty and complexity. If we wish to arrive at the whole truth of the matter we are bound to look at causation from every possible point of view. This is no easy matter when preconceived opinions are apt to beset us on every side.

I cannot do better than conclude by quoting a passage *a propos* of these remarks, taken from a paper on "Pandemic Influences", by Inspector-General Lawson, published in the *Army Medical Blue Book* for 1861. He says—"The advance of knowledge has been much impeded by the disposition, so strong among medical writers and observers, to regard disease as they individually see it, and refer its prevalence and character to circumstances affecting obviously the population within their immediate sphere; which, had their observations been extended to other countries, a similar prevalence of disease might have been found under local circumstances very different from those thought so essential to its occurrence among themselves."

ON CERTAIN VIEWS REGARDING FEVER IN INDIA.

By Surgeon-General C. A. GORDON, C.B., M.D., etc., etc.,
Army Medical Department.

[*Read: April 29th*, 1880.]

Mr. President,—Within the past few years the subject of Fever, as affecting British troops in India, has been considered from a point of view altogether different from that according to which it had been previously investigated. Hence it happens that considerable divergence of opinion exists between the older observers and the more recent with regard to several important details embraced in that inquiry. In the meantime this very serious statement is officially made, namely, that "of late years the proportionate fever mortality has increased nearly a fourth, and the proportion of fatal cases entered as 'enteric' fever to all other fevers has quadrupled". It accordingly appears to me that further investigation may be facilitated and helped on by means of a short summary, such as I now have the honour to submit, of some views expressed with regard to history, causation, phenomena, pathology, and a few other circumstances connected with the large class of disease embraced under the general head of *Fever* in that empire.

In my attempt to bring together each set of views, and compare them with one another, I have considered it expedient to refrain from introducing the names of the authors of those opinions. By adopting this plan, I hope the more distinctly to indicate that I deal simply with *opinions*, not *persons;* for where views are held, of a nature absolutely divergent from each other, and conflicting against others, such as I am about to narrate, the expediency, I think, becomes manifest of avoiding any expression of a nature likely to introduce an element of personality into our attempt to throw light upon a very difficult question, in a science which by its very nature can hardly be described as *exact*.

It appears desirable that I guard myself by one more preliminary remark, namely, that the several views I am about to express are those of eminent and well-qualified

observers on either side of the question. All that is *mine* is contained in the *deductions* I have drawn from a comparison of those views; and how far those deductions are correctly arrived at, or otherwise, you, sir, and this meeting will be in a position to judge as we proceed.

1. *In Ancient India.*—The hakeems, or native physicians of India, recognising the close alliance existing between fever and dysentery in that country, assign both affections to similar causes, namely, high temperature, humidity, exposure to changes of weather, fatigue, and privation; and the circumstance is, to say the least of it, interesting, that among the views at the present time entertained with regard to that form of fever to which I am about more particularly to solicit your attention, namely, that attended by ulceration of the iliac glands, with or without other intestinal lesion, is, that its alliance is more with dysentery than to idiopathic fever. Thus is the theory of twenty-four centuries ago reproduced in our day. In ancient times works on Hindoo medicine contained descriptions of thirteen kinds or forms of what were called by the early physicians "mixed fevers", the following being the characteristics, according to them, of the *second* form in their enumeration, namely, "*Pittulbana* or *Asukari.* The peculiar symptoms of this form are looseness, giddiness, fainting, aphthæ in the mouth, red spots, and a burning sensation over the body". (*Hindu Medicine*, by Dr. Wise, p. 228.)

2. *By later Writers.—a. The Older.*—With more especial reference to fevers affecting British troops and European residents in India, the older medical officers wrote as follows, namely: In the course of almost every protracted fever which terminates fatally, there is a period marked by subsultus tendinum, rapid and feeble pulse, low delirium, brown sordes on the tongue and teeth, usually considered *typhoid*—that is, in regard to *condition*, not to specific nature of the disease. Certain organic lesions were described as *results* of endemic fever—not as of themselves constituting the primary disease itself; it was stated that the more irregularity intermittent fever presents, the greater is the derangement of the abdominal viscera, including liver, spleen, and alimentary canal. Intermittents often run into remittents, and then become continued; remittents and continued fevers in their turn change to intermittents. The expression *typhoid*, as indicative of a *condition*, was in general use in reference to these several forms of endemic fever, the fever itself believed to be solely due to endemic and climatorial influences acting upon par-

ticular diatheses and individual states of body and constitution. In the classification of agues the *third* variety was that "with exhaustion of the powers of life, or with *typhoid* symptoms"; the fourth associated with disease in some internal viscera. In the classification of *remittent* fever the fourth variety was "the malignant, or remittent with *typhoid symptoms*". Mark the expression—typhoid *symptoms*. Of *continued* fever three varieties or forms were enumerated—namely, 1. The inflammatory; 2. The bilious inflammatory; and, 3. The malignant, *or adynamic*. The circumstance was recorded that "in the more malignant kinds of fever the spleen is often observed enlarged and softened; the duodenum, jejunum, and ileum, especially the duodenum and termination of the ileum, are frequently diseased in their mucous surface, which is inflamed in patches". "In several instances the ulcerations (at the termination of the ileum), which sometimes are large and far apart, at other times small and agglomerated, have nearly or actually penetrated the tissues of the intestines. Morbid appearances such as these are generally found in cases of fever—that is, *endemic* fever—in which diarrhœa and dysentery *supervened* (observe the expression *supervened*), and which in their progress assumed an *adynamic* or malignant form."

b. More recent.—Still more recently, we have the views expressed by medical officers who have enjoyed ample opportunities of comparing the opinions already enumerated, and of considering, in reference to them, individual cases and more general outbreaks occurring within their own cognisance. Among the views thus formed are the following, namely: "When agues and remittent fevers in India terminate fatally, unless that happens by sudden supervention of cerebral symptoms, to which the term heat apolexy is usually applied, it is by their running into the continued type, and assuming symptoms to which the designation typhoid is usually given." "In fact, the tendency seems to be—to return all cases of fever that die as cases of enteric fever, and those that recover as examples of agues, continued, or remittent fevers." "In all deaths happening early in the disease, cerebral effusions, congestions, etc., will be found to predominate; whilst, in those occurring later in its course, it will be the enteric (that is, intestinal) lesions that are the most marked." On these points, it appears desirable to remark that the conclusions thus expressed occur in more detailed form in the reports before us, and are the result of inquiries officially conducted by a highly qualified medical officer, viz., "That the expe-

rience of British troops in India does not support the conclusion that it (enteric fever) is an affection dependent for its propagation on poisoning of a specific fæcal character", although the circumstance nowhere meets with denial, for the reason, apparently, that it is undemonstrable that under certain circumstances, a specific fever, having the main characteristics of typhoid or enteric in Britain, may owe its origin to pythogenic causes. This may be so, notwithstanding that of upwards of 175 cases, details of which are given in the two special reports before us, in not one was the disease so named traced to such a source, even by the medical officers reporting them.

With reference to cases of fever, resembling in their phenomena those of the specific form just alluded to, the question has been asked—Is this the same disease which might be contracted in London, Dublin, Windsor, or elsewhere, from a water-closet, drain, sewer, well, or milk can? and, as a reply, it has been stated that "a form of fever, exactly like European typhoid, except in its etiology, exists in India, and other hot and malarious countries; and that it is due to climatic causes, not to filth or specific causes, such as give rise to it in England and elsewhere". "Fevers of malarious (or climatic) origin may be, and often are attended by such bowel symptoms as diarrhœa, hæmorrhage from the intestines, ulceration of the mucous membrane of the large and small intestines, etc.; yet in them, every attempt that has been made to trace the origin of the disease to a specific pythogenic source has failed; they generally occur in climatorial conditions of a special kind; they run a course of extreme irregularity; it is only in abdominal symptoms and pathology that these cases really resemble genuine enteric fever; the head symptoms, as a rule, differ widely from those of enteric fever." The medical officer who thus expresses himself as to the circumstances which qualify him to form an opinion, says: "I have seen a great deal of genuine enteric fever in North America and in England. My experience of Indian fevers hardly amounts to a period of four years, but the time I have doubted the nature of these so-called enteric fevers is coextensive with my service in India, and may perhaps weigh fairly in comparison with a longer period of unquestionable acquiescence. No evidence exists of the disease having been imported from England, or from one station to another, nor of its spread by contagion; neither were the water-supply, nor any article of food or drink at fault; but the mere existence of enteric is evidence of insanitary

conditions." Such is the statement made in official reports. Such the standard adopted.

c. Present views.—Within the last twenty to twenty-five years other views with regard to the nature and etiology of fever in India have been expressed. It has been asserted that "the great prevalence of typhoid"—that is, of a specific form of fever so named, as distinguished from typhoid, as indicative of a *condition* occurring in the course of endemic or climatorial fever—" was long concealed from view behind the forms of fever which were denominated malarious; everything was at one time jungle fever", as the severer attacks of remittent fever used formerly to be popularly called; "whereas it is now perfectly well known (mark the decided nature of the expression) that a great part of the so-called jungle fever is neither more nor less than common typhoid", that is, the specific disease so named. In reference to climatic and other endemic influences, the effects of which have been acknowledged since the date of the earliest Hindoo writers on medicine, the opinion has, even within the last five years, been expressed that "among the most serious errors"—such is the expression applied to the views of all previous writers—"is the assumption" (listen to that,—assumption) "that there is a fever of the country at all—that is to say, a fever due to climatic influences, or to natural emanations from the soil, and not to conditions artificially produced by the inhabitants themselves"; the conditions thus alluded to being such as are usually comprised under the heads of defective conservancy. Can it be that the views thus expressed are those of a writer who, in his own person, has had real experience of an Indian climate? Have not the views rather an impress of home manufacture upon them? It has been said that "of late years enteric or typhoid fever has been recognised as a special cause of mortality in the British army serving in India". "It is not at all probable that high temperature will cause enteric fever, but it may so act in the body as to render the effect of known existing causes of enteric fever more intense than it would otherwise be." "The removable causes enumerated are the introduction into the system of putrescent fæcal matter. There is evidence that Indian climates, acting on young constitutions, predispose to febrile action; but there is no evidence to show that climatic influences will cause enteric fever; while, besides climate, the young soldier has to contend against local causes, which are well known to be the exciting agencies of the disease. The disease is officially described

as a filth fever. All that is required in reference to the above is a definite expression of what is intended to be implied by the name enteric, as applied to a form of fever; but this is precisely what does not appear.

These views have been very distinctly expressed by high sanitary authorities, after this manner:—"The recurrence of the typhoid fever at any station should lead to immediate inquiry as to the state of cleanliness of ground in the vicinity likely to act injuriously on the purity of the air; as to the condition of latrines and water-courses, and the condition of the bazaars frequented by men who have been attacked." All excellent precautions, but altogether passing by such conditions as obtained the notice of medical officers while the filth theory was only considered in relation to the general objection against neglect of cleanliness, and of conservancy. In those days, such circumstances as the following were looked for as causative of fever in India and the tropics generally, and hygienic measures taken accordingly; namely, in regard to youth, habit of body, temperament, exercises and duties, habits, exposure, over indulgence, and others affecting the individual; at the same time that climatorial, seasonal, and endemic influences were recognised and acknowledged. (Within parentheses let the observation be made, that these opinions here described at of the present time, are in all instances taken from the works of recognised writers and authorities on the subjects to which they refer, and from reports by official authorities. What, then, was my surprise to find this query, by a high officer, to whom this manuscript was lately submitted: "Are not these the past views?" They most assuredly are the very latest views to which I have had access; and I can now only ask the question, "If they really are the past, what are the newest new, and of course improved views, to make room for which they have been relegated to such limbo?" Do not the charts in the second report before us indicate by their zig-zags of fever lines that, while in the mass such lines show a tolerably even quantity of that particular class of disease, their individual variations occur in such a manner as, to say the least, is very remarkable, either with regard to a name, or to the actual nature of the disease to which a name is applied.)

Here is still another view regarding fever in India, so recent indeed as to have only reached us by last mail. A medical officer, whose name is given, believes that a new disease has appeared in the epidemic, at the time he wrote, in Calcutta. He believes that it is probably a description

of hungerpest, always traceable, more or less directly, to antecedent famine or scarcity, and susceptible of identification with both the continuous low yellow fever of Cormack, and the hæmogastric fever of Copland. He claims for some of the manifestations of the new pestilence all the quasi-typhoid symptoms which have occasionally been observed of late in that city; uncompromisingly denying that there is any real typhoid in Calcutta just now at all. Miasmatic poisoning from drains may, he says, be detected, doubtless; but if the symptoms of the attacks in which this causation was suspected were otherwise irreconcilable with the ordinary diagnosis of typhoid fevers, it is more reasonable to assume, generally, that other maladies may have been intensified for the time by the epidemic, than that extraneous symptoms, irreconcilable with certain diseases, were actually caused by them.

d. Deductions.—Such are some of the opposing views enumerated on the present subject, and they might be added to, did time permit. Among the questions they suggest are these:—How far do the phenomena recorded correspond with those that characterise fever purely climatorial and endemic in its nature? How far those that occur solely as a result of reception into the system of a specific poison contained in decomposing human fæcal matter? Very important issues attach to the manner in which these questions are answered. First and foremost is the particular line of treatment to be followed according to the theory of causation; then there are the means of prevention, involving, as these do, questions of public finance in connection with the destruction or reconstruction of buildings and their several appliances.

3. *Standards adopted.*—The circumstance becomes evident, during investigation of the subject, that various standards of what constitutes typhoid or enteric fever, have of late been adopted in India. Thus, according to one, the phenomena so designated are not specific in their causation or nature, but are mere accompaniments of endemic fever, whether continued, remittent, or intermittent. According to a second opinion, they do not arise from the operation of such influences, but from the presence of filth in air, earth, water, or food; when the presence of filth cannot be demonstrated, it is assumed. According to a third opinion, the presence of intestinal, that is enteric, complication in the course of any variety or form of fever, irrespective of the manner of causation, constitutes enteric fever, apparently

in the sense that the terms pulmonic, hepatic, or cerebral, might be used, in a specific sense, to a case of fever in which either of those complications was present. It is further on record, that the terms typhoid and enteric being used synonymously, the result has come to be that the former so-named condition, when present in a case of fever, is held to authorise the application of the term enteric, even in the absence of intestinal complication; and, on the other hand, the presence of intestinal complication, that of typhoid, even in the absence of a low adynamic condition to which the term was formerly restricted. In a few instances the presence of a cutaneous papular rash, rose or pink in hue, occurring in the course of a fever, was held to justify the application of the term enteric. Thus the question naturally presents itself: Wherein lies the difference between the type of fever above alluded to, and those in former years described as continued, remittent, or intermittent, with a low or typhoid condition of the patient, with or without intestinal complication; or with intestinal complication, with or without a low typhoid condition of the patient? Are not replies to such a query given by the eminent authorities quoted in the reports before us, namely: "Ulceration of the Peyerian patches is by far the most noteworthy single fact, pathologically speaking, in the natural history of enteric fever, yet it will not do on a fact like this to found in the matter of diagnosis; after you have settled that there are cases of fever with ulcerated intestines, you have still to settle whether these cases are different in nature from other cases, in many respects similar, in which the intestines are not ulcerated." And again: "Admitting that fever shows itself under various forms, the effect upon the mind of all this subdivision is bad and hurtful. It encourages a disposition, already too prevalent, to prescribe for a disease according to a name. There is no genuine distinction between continued fevers that can be relied on." A still more recent author writes after this manner:—"Continued fever frequently occurs without any organic lesion; in other cases, cerebral or pulmonic complications might furnish a specific name." He further adds: "I agree, therefore, with those who consider fever as an essential disease, dependent on some unknown constitution of the blood, and occasionally accompanied or followed by various local lesions of the cranial, thoracic, or abdominal viscera, and with various eruptions of the skin."

A sanitary authority in England writes:—"The fact is, unfortunately for the statistician, the expression typhoid

fever is not always used in the same sense by different medical men, but is occasionally applied to cases to express a generally low typhoid condition, without the specific symptoms of enteric fever being necessarily present."

Does not, therefore, the manner itself in which the terms enteric and typhoid are synonymously applied leave scope for want of precision, such as records indicate to attend their use in India?

As a commentary on the above remarks, the following occurs: "Cases of diagnosed typhoid with Peyerian ulceration would be classed with other fevers having ulceration as a 'complication' under the same head of 'enteric' fever." Here there appears to be a standard adopted from the mere existence of "Peyerian ulceration", even occurring in endemic and climatorial fever. But how about cases called enteric, where no intestinal symptoms are manifest? How about cases of endemic fever, in which cerebral or pulmonic, or hepatic complications are present, intestinal absent? Are they to be severally named in accordance with the existing complication? How about cases of "enteric" fever in persons beyond the age during which the presence of Peyer's glands exists? How about cases of fever running into a typhoid, low typhous—not typhus—condition, but without organic lesion of any kind? We know, as a result of observations, that organic complications occur not necessarily at the commencement of fever attacks in India, but at subsequent stages of its progress.

4. *Causation.*—Among the assigned causes of specific typhoid or enteric fever in Britain and Europe generally, the following are enumerated. 1. *Emanations from sewers.* But although, in many cases, it does so, this result is not invariable; exposure of this kind may be followed, not by specific typhoid fever, but by nausea, faintness, and diarrhœa, these symptoms ceasing on their subject being withdrawn from the emanations. Among other diseases owning the same cause are diphtheria, erysipelas, plague, cholera, dysentery, yellow fever, etc.

2. *The Use of Impure Water.*—But so also are other diseases, more particularly intermittent fever, diarrhœa, cholera, and dysentery. But, on the other hand, at certain places where the water is notoriously contaminated, residents have an immunity from enteric fever.

3. *Fœcal Contamination of Air.*—That this cause operates in producing specific *typhoid* or *enteric* fever in Europe and in America appears to be placed beyond a doubt by the direct evidence adduced on the point. But such causes,

when they exist even to a great extent, are not always thus operative, as in the case of air rendered impure by decomposing bodies, putrid meat, knackeries, tanneries, and so on.

4. It has been produced by violent and continuous military exercises and the exposure connected therewith; also great cold after such exercises. According to this acknowledgment, a train of phenomena in all respects similar to those that result from a specific cause, by some writers considered to be *a poison*, are admitted to owe their causation to conditions of the body itself, and to those of the surrounding atmosphere. And yet the power of other atmospheric conditions, such as exist in India, also to cause the like phenomena is denied, as we have already seen. Is it not the case that even here in England "all professional men are not yet agreed as to the real cause of enteric fever; also that the causation is so little understood, that it gives us no help in the diagnosis"? But with regard to a form of fever, the definitions of which vary among themselves, and the causation of which is still undecided, so long as we depart from generalities and individual conditions, inquirers in India whose inquiries have failed to demonstrate certain specific causes of what has been indicated to them as a specific disease, have had very severe expressions applied to them. Meantime, however, their views are leavening opinion at home.

a. Commentaries.—In reference to this part of the subject the following extract from remarks on enteric fever by a well-known English author seem appropriate. He had previously held to the doctrine of an imperative necessity for the specific poison being imported into the body. His subsequent experience, from certain seeming illustrations and circumstances, has gone to confirm the belief that under special and peculiar conditions enteric fever may become instituted in the system without any external agencies other than such common causes as give rise to that lowered tone of vitality which favours the springing up within itself of the morbid phenomena in question. According to another equally eminent authority, "specific causes are not in all cases required in order to produce an attack of fever; changes may take place spontaneously in one or more of the functions, and proceed to give rise to the worst forms of fever". "That such is the case", he adds, "appears to be fully proved; also, that the existence of the condition described as *epidemic constitution* is important in connection with the causation of fever."

The best instance of the inefficiency of sewage decomposition to produce enteric fever is that of the Thames in 1859. "For the first time in the history of man the sewage of nearly three millions of people had been brought to seeth and ferment under a burning sun in one vast *cloaca* lying in their midst. Stench so foul as never polluted this river air; river steamers lost their accustomed traffic, and travellers pressed for time made a circuit of many miles rather than cross one of the city bridges." With all this condition of the Thames the health of the metropolis was remarkably good, *and enteric fever actually less than usual.* Is it therefore to be wondered at that the reporting medical officer should thus express himself. "As regards enteric fever, and probably other contagious diseases—to wit, diphtheria, *we do not think that the filth and drain theory will stand.*" Please to observe this is not my language; it is that of an eminent English physician, and leading authority in regard to our present subject.

While the present paper was in course of preparation, a communication to you, from a recognised authority on the subject of fever in Britain, was through your courtesy placed before me. The writer of that communication wrote thus:—"I have devoted some time to the study of the etiology of typhoid fever, and I have come to the conclusion that the worst cases in which it is ascribed to filth, it is in most cases wrongly so ascribed. I do not believe that there is any necessary connection between filth and enteric; and, indeed, I doubt if there be any at all. At the same time, I would not like to say that any kind of filth might not contain enteric poison. The filth theory is really due to a name; and, after a very anxious consideration of the evidence in favour of it, I have come to the conclusion that there is really none worthy of the name. The same may, I think, be said of drains, milk, and water supply. Some of the most recent of these are ingenious, and to *devout* believers, no doubt, satisfactory; but I am heterodox enough to doubt the whole thing." "I think you maintain a climatic origin—that is, Trousseau's view, age, climate, and overcrowding, and I don't think that we have got beyond that yet."

b. Results in Madras.—In Madras, pythogenic causes are for the most part non-existent; and in the few in which they are present, inquiries show that they were inoperative. Thus in only a very small number of stations do sewers exist, and where they do, no case of *specific* fever has been traced to their presence. Diarrhœa and dysentery are by

no means seldom traced to the use of water contaminated by decomposing animal matters, and malarial fevers to the use of waters in certain districts similarly contaminated by decomposing vegetable matters. At certain Hill Stations such a connection between a particular kind of diarrhœa and water rendered impure by fæcal matter was acknowledged to exist; but of the whole number of cases recorded in the reports before us, as a matter of fact no such connection was demonstrated as existing between the cases recorded as of enteric fever and contamination of the water. As regards contamination of air by fæcal matter at one station, where an outbreak of so-called enteric fever took place, the troops occupying a range of barrack rooms side on to, and more directly exposed to emanations from latrines, suffered in a less degree from the prevailing disease than did those in barrack rooms placed end on to the same latrines, and also to the prevailing winds. In the one case there was through and through perflation; in the other the ventilation was less complete; in both, the soldiers were, for the most part, young lads, brought rapidly from Ireland to the hot damp climate of Malabar, at a season when malarious fevers were unusually present even among the native population. In the second instance, the atmosphere of the place was extremely offensive at times, yet no case described as "enteric" was recorded during the period embraced by these reports. According to replies by medical officers, in no case was so-called enteric fever actually traced to food or milk used by them; nor must the circumstance be lost sight of, that while soldiers' wives and children, who use bazaar milk in large quantities, enjoyed very great immunity from the disease so-called, the young soldiers, who used comparatively little milk, furnished the bulk of the cases. As to the disease being caught in the bazaar from the use of contaminated milk or water, it only needs to be observed that the purpose for which the British soldier visits a bazaar is other than to indulge in milk and water.

5. *Phenomena.*—A particular train of phenomena is held to characterise specific enteric fever. But according to works on the subject referred to in the Reports before us, such phenomena are not always present in their entirety in the specific fever itself, but some or all of them are so in the course of fevers in India distinctly due to climatorial and endemic causes, and also in some other diseases than fever, both in that country and in this. *Tympanitis* occurs not only in specific enteric fever, but also as a serious complication in a variety of diseases. Abdominal tenderness

in fevers from non-specific diseases, gurgling in the right iliac region in diarrhœa, also from non-specific causes, end in dysentery; diarrhœa, absent in eleven cases out of sixty-two of specific pythogenic fever, occurs in fevers of non-specific origin, climatorial and otherwise. In the *jail* distemper, which prevailed in the middle of the eighteenth century in England, "an offensive diarrhœa brought numbers to their end". Hæmorrhage, by no means constant in occurrence in specific enteric fever, takes place in adynamic or typhoid dysentery; in the more dangerous seizures of remittent fever in tropical countries generally; in cases of inflammatory fever, formerly known as *synocha*, and more recently as continued fever; and so, epistaxis and intestinal hæmorrhage are recorded as occurring in the course of fever attributed to the use of tainted veal and sausages.

6. *The Temperature* line in typhoid fever is said to run such a typical course, with regular diurnal variations, with so steady a rise to the fastigium, and decline to the defervescence, that from a mere inspection of the chart, we can generally diagnose the disease. As a matter of fact, in three so-called typical charts, extracted severally from the works of three recognized high authorities, the line markings differ from each other absolutely in character; in those having reference to cases of so-called enteric or typhoid fever in India, fac-similes of which are given in the second report on the table, their several characters are so various that it does not appear practicable to draw conclusions from them. One of the older Indian medical officers, many years ago observed, with reference to temperature in endemic fever of that country, that "it is well to distinguish between *heat* or symptom, and the *disease* fever". Is there not reason to believe that temperature, in a case of fever said to be enteric, is, at the present time, by some observers at least, alluded to as if it were itself principally, if not altogether, the disease, instead of being one among others of its phenomena?

7. *Cutaneous Eruption.*—According to a number of English writers, the presence of a specific eruption is considered to be characteristic of enteric or typhoid fever. Some also believe that such eruption is found in no other disease except enteric fever. A very eminent physician and writer observes that in four years he never saw a single exception to the rule that rose spots are diagnostic of typhoid fever, that is to say, of an affection having for its anatomical character disease of Peyer's patches. That is the

standard of the disease so called. On the other hand, according to reports before us, a very eminent French authority, while disagreeing with those who hold that such eruption is specially characteristic of the disease, nevertheless considers this eruption of very great importance in its symptomatology. In Paris it is said to be always found, while in some other cities it is entirely wanting in different epidemics. Even in the same epidemic it is not always present. Rose spots occur in other forms of fever than specific enteric; in America their presence is recorded in cases of purely malarial fevers. In England, a well qualified author writes thus regarding enteric fever: "If you rely on the characters prominently given, and especially with regard to the eruption, it must be evident you will be frequently deceived." An eruption, similar in character to that of specific enteric fever, has been recorded in connection with sausage poisoning and trichinosis. With regard to India, we find the circumstance recorded that the presence of eruptions on the bodies of men suffering from any form of severe disease during the hot weather is the rule, and it is during the autumn months that the most serious forms of fever occur. In the Reports before us, we find various forms of eruptions noticed as occurring in fatal cases of fever, said to have been enteric, and also in those that recovered; some of the eruptions being severally described as typhoid, rose eruption, lenticular spots, characteristic, suspicious spots, measly eruption, and so on. In the first of the Reports on the table there occur details of fifty-four fatal cases of so-called enteric or typhoid fever. In twenty of that number an eruption of some kind was noticed as being present; in twenty-seven no eruption was present; and in seven, particulars are not sufficiently detailed to render it clear whether an eruption was or was not present. Of the twenty cases in which an eruption was present, ulceration of Peyer's glands existed in eleven, or 55 per cent.; ulceration did not exist in three, or a ratio of 15 per cent.; and its presence or absence was unrecorded in six, equal to 30 per cent. In the twenty-seven cases in which no eruption existed, ulceration of Peyer's glands was present in sixteen, or 59 per cent; there was no ulceration in eight, or a ratio of 29 per cent: in two cases, or 14 per cent., ulceration affected the ileo-cæcal valve alone, and in one case, equal to 7 per cent., no record appears as to the presence or absence of ulceration. In the seven cases in which the presence or absence of eruption of any kind is unstated, ulceration of Peyer's patches was

present in four cases, or a ratio of 57 per cent.; not present in one, or 14 per cent., and unrecorded in two, equal to 28 per cent. So far, therefore, as these particulars indicate deductions, they are, that in the cases of Indian fever referred to, no special connection was traceable between the presence of cutaneous eruption and the existence of ulceration of Peyer's glands; also that the cutaneous eruption, when present, was different in its character in different cases. Is not then the conclusion to be drawn from these particulars, that with regard to the cases, details of which are given, neither is the presence of an eruption to be held as distinctive of specific enteric fever, nor necessarily of the existence of ulceration in the aggregate glands of the ileum?

8. *Duration of Attack.*—Of a series of cases of enteric or typhoid fever in England, the duration of the disease was 12 days in 1 case; 14 to 21 in 19; 22 to 28 in 40; 29 to 30 in 9; above 30 in 6. In the outbreak at Clapham, some cases were fatal in less than four days. According to a high authority on the subject, the average duration of the disease is twenty to thirty days. Of 255 cases recorded in Massachusetts, the average duration was twenty-two days; according to another authority, the average in the same province was thirty-nine days. How far in such instances the doctrine of averages is really suitable may be a question; but as averages are drawn, such are the results indicated by them; the results being that no definite rule can be said to hold in regard to the length of time a case of typhoid fever, as at present defined, may be expected to continue. Of the 175 cases related in the first of these Reports on the table, the following is a summary of results regarding their duration, namely, first, with regard to those in which recovery took place, the duration of the attack in one was seven days, in another 105; between these limits there was great variation, according to station, but for the purposes of this paper they are considered in the mass, and according to the order followed above. Thus, in three cases, the duration was under fourteen days; in 4, between 14 and 21; in 8, from 22 to 28; in 5, from 29 to 30, both inclusive, and in 72, above that period. In the mass, the average duration, arithmetically expressed, was forty-five days; but at different stations, the averages varied thus: 11 at Kamptee; 24 at Thyet Myo; 38 at Cannanore; 40 at Wellington, a hill station; 43 at Maliaporam; 55 at Bellary; 56 at Bangalore; 60 at St. Thomas's Mount, and 84 in the Andamans. With regard to the fatal cases, two occurred

in one day of illness: 2 in 2; 4 in 4; 1 in 5; 2 in 6; 4 in 7; 3 in 8; 1 in 9; 4 in 10; 1 in 11; 2 in 12; 1 in 14; 3 in 15; 2 in 16; 2 in 17; 1 in 18; 1 in 25; 1 in 29; and 1 in 35; thus giving an arithmetical average for the whole of nine days. A similar analysis of details contained in the second Report indicates, with regard to the eight cases in which recovery took place, that the limit of duration ranged from eighteen to eighty-six days; that one was within the period, as above, of fourteen to twenty-one days; one from 22 to 28; one from 27 to 30; and six beyond that period: the general average of all, thirty-four days. Of the eight fatal cases there recorded, one occurred in two days of the attack; 1 in 9; 1 in 10; 1 in 18; 1 in 19; 1 in 34; 1 in 36; and 1 in 68; general average, 24. In the several classes of cases enumerated, the nature of illness is considered to have been endemic and climatorial; in them the variety in persistence was equally great as in specific enteric; nor must it be omitted to be observed that in this respect locality, and, no doubt, treatment, had their influence.

9. *Pathology.*—Induration, and at a later period, ulceration of Peyer's glands and solitary glands in the ileum, are described as constant in, and peculiar to, enteric fever; and as constituting "the anatomical sign of typhoid fever"; also that such conditions may extend to the solitary glands in the cæcum and ascending colon. This ulceration is described as specific in character, and even as constituting the special intestinal eruption of specific enteric fever. But other authors are of opinion that pathological conditions undistinguishable from those which occur in specific enteric fever, occur also in the course of endemic fevers more specific in their nature; and in this sense they were noted by the older medical officers in India in connection with intermittent and other forms of endemic fevers. It is asserted that the typhous, that is, typhoid process, is purely artificial; that in a case of typhoid or enteric fever, the ulcers in both small and large intestines were undistinguishable from dysenteric ulcers; that precisely similar conditions occur as results of poisoning by arsenic and cicuta virosa, poisonous mushrooms and colchicum; that they have been observed also in typhus; that they occur in cases of ordinary climatorial fevers in India, in cases of protracted cholera, and in enteritis; that in such they are no more than incidental occurrences. That such lesions in the cases alluded to are mere incidental occurrences is indicated, according to some writers, by the circumstance that they only take place

in the advanced stages of the fever, not in the early stages; that a disease having all the characters of specific enteric fever occurs in persons of an age beyond that when Peyer's glands exist; and, moreover, that such changes also occur in adynamic and malignant fever, even to a greater degree than in enteric—particularly such as are of malarial origin; a similar result takes place in hectic fever. What was formerly designated glandular enteritis, was described as taking place consequent upon fevers, or during convalescence from them; also as "endemic in some countries, partly owing to high temperature in connection with humidity of the atmosphere, partly to miasmata". Such changes were said to occur in fever generally. Lesions of the same kind, occurring in the same organs, are more frequently met with in hot climates than in temperate; in these the mucous membranes, Peyer's and Brunner's glands, the brain and lungs, are chiefly affected. With reference to Peshawur fever, it is recorded that experience has taught that anatomy is not always to be trusted as a guide to the cause of death; that very similar textural changes are often produced by widely different systemic diseases. A very eminent physician thus wrote in 1832: "Morbid appearances found after fatal fevers are often observed to be remarkably various, even in cases the leading symptoms of which are nearly the same, and they are far from bearing any fixed proportion to the intensity of the symptoms of affection of the parts in which they are found." An army medical officer, of great and varied experience, wrote thus with regard to the morbid appearances in fatal cases of yellow fever: "Slight ulceration of the lower parts of the ileum; severe ulceration of the large intestines, as in chronic dysentery; perforation in the lower part of the ileum, large ulcers in the same part; small perforation in the upper part of the ileum; Peyer's patches enlarged; several deep ulcers in the lower part of the same intestine; the lower part of the ileum studded with ulcers." In continued fever, non-specific in character, the same writer enumerates small ulcers in the ileum, cæcum, and colon; spleen soft; numerous small ulcers scattered through the ileum.

From the data thus summarised then, I hold that the conclusions expressed in the Reports on the table are supported by evidence; namely, that pathological conditions, now stated by some writers to be characteristic of, and confined to a fever resulting from a specific cause, namely, filth, exist also in fatal cases of various forms of disease,

specific and non-specific, occurring in tropical and sub-tropical countries; and that, therefore, in such instances, to apply to the disease in which they occur a term having reference to the existence of a particular condition, as resulting from non-specific causes alone, is inappropriate and misleading.

10. *In relation to Season.*—According to former writers, thirty-five years ago there was no evidence of intestinal disease in the continued fevers of London, except in autumn. Most of the outbreaks in English towns and villages, during the last thirty years, have occurred in autumn; and the belief is entertained that the autumnal fever of last century, in Britain and Ireland, was the same as that to which the name enteric fever is now applied. In the colder months of the year, there is greater risk of inflammatory complications of fevers; in autumn we look more for diarrhœa or dysenteric complications engrafting themselves on the disorders; when the air is close and foul, the symptoms show a much greater tendency to the typhoid type.

Turning to India, official reports state that in 1872, "the occurrence of enteric fever in India was clearly related to season". "Between the middle of August and end of September, cases occurred at stations all over India; and they were accompanied by other cases of purely climatic nature." As in this country, therefore, season is admitted to influence the occurrence of fever attended by bowel complication, so the above-noticed circumstances render the fact evident that in India a similar rule holds good.

11. *Climatic Influences and Malaria.*—Inasmuch as the *fauna* and *flora* of localities are affected and modified by climatorial influences; as the constitution of man is similarly affected, so it is admitted by medical officers and others who travel extensively, that the phenomena of disease are thus modified. According to them, "each country has its own particular type of fever, depending, more or less, upon physical causes, and requiring certain modifications in the method of treatment". According to others, infraction of Peyer's glands is endemic in some countries, partly owing to high range and daily vicissitudes of temperature in connection with great humidity of the atmosphere, and partly to miasmata. In India, at a particular station, during the same season of the year, among the same body of men, under similar circumstances, different forms of fever at times prevail, namely, intermittent, remittent, and continued. Such forms are by certain medical officers looked upon, under such circumstances, as one and the same disease, dependent upon the effects of heat,

moisture, atmospheric electricity, and ozone. All forms of fever in India, under certain conditions of person, climate, and locality, become adynamic, low, or typhoid, whether at the most they are intermittent, remittent, or continued; also, in that adynamic state the danger to life is the greater, in proportion to the extent of affection of internal organs, whether abdominal or cerebral.

The circumstance is well known in India and other tropical and semi-tropical countries, that the term malaria, besides signifying marsh miasm, is applied to otherwise undefined influences, existing in localities and in geological formations incapable of yielding miasm of that nature; also that in such localities and on such geological formations, forms of disease in all respects similar to those which occur where miasms prevail, also present themselves. Among them are hepatitis, dysentery, remittent and intermittent fever, and so on. Hence, in India, the expression malaria can only be accepted as a synonym of climatic influences. Even the chief advocate of the pythogenic theory of enteric fever admitted that there exists an analogy in many points between it and diseases acknowledged to be malarious. The occurrence of malarious fever in epidemic form at Indian military stations is acknowledged as a fact, and especially among the more recently arrived; also that the type is rendered more severe, according to the unhealthiness of the station, and of the season. In them, according to former writers, "the visceral disease ought not to be viewed as the immediate cause of the febrile excitement; or, in other words, that the fever is merely a general disorder supervening to a disease of a particular organ; on the contrary, the exciting causes of fever produce disorder of the frame generally, which, owing to the predisposed state of certain viscera or textures, occasions a permanent derangement in them, and as we have just found it asserted, such a predisposition or tendency exists in the glands of the ileum. The effect of influences of this nature was thus acknowledged by the Royal Commission on the state of the Army in India, 1863, namely:—"By far the larger proportion of mortality and inefficiency of that army has arisen from endemic diseases, and notably from fevers, diarrhœa, dysentery, and diseases of the liver; the predisposition to these diseases is, in part, attributable to malaria, in conjunction with extremes of temperature, moisture, and variability"; that is, to local and climatorial conditions. Nor is it alone upon man that such influences exert their action. Thus, in the Rohilcund terai, in the valley of Dhera Dhoon, herds of domestic cattle are, from time to time, carried

off by deadly fever, and are, for the most part, subjects of splenic disease and cachexy. Imported animals into India suffer from endemic diseases, and die by them, in even equal, if not, indeed, greater proportion than does man; imported plants droop and die, or become, to a greater or less extent, changed in their characteristics. In the very latest official report available while preparing this paper, the following remarks occur: "If the facts point in the direction of any efficient predisposing cause, it is to newness of climate and malaria, acting on new arrivals and immature constitutions." "After 20th April, in any locality, too much care cannot be taken of new regiments; for it is in this week that exposure is very certain to be followed by the appearance of fever; and again, the tendency for the fever to reappear towards the close of the monsoon season must be kept in mind." "The fever is chiefly due to climatic conditions, acting on special constitutions."

Is not, therefore, the fact rendered evident by the above, that the pythogenic theory of fever in India has been abandoned by the authority referred to, also that the endemic and climatorial has been accepted in its stead; in other words, that the views entertained by the older medical officers have once again been received into favour? This being so, where is the propriety of retaining a name applied in accordance with a theory now found undeserving of continuance.

12. *In relation to age.*—In England, the age of greatest liability to enteric fever is 15 to 25; the next from 25 to 30. But a case is related in which a child died of the disease so named on the eighth day after birth, although its mother had not the disease, either during pregnancy or after confinement. Fatal cases are recorded in persons aged 60, 75, 83, and 87 years. Thus, on the one hand, such cases occur before the aggregated glands have attained their full development; on the other, after that in which their activity has ceased. In India, the relation of endemic disease generally to age corresponds pretty closely to that of typhoid fever in Europe. The circumstance that mortality from all such diseases fell heavier on the young and recently arrived soldiers than on the older ones, was often recorded by the medical officers in former times; young soldiers, in larger proportion than ever, are being sent to India, and the high rate of mortality among them, not alone by fever with intestinal complication, but by fever without such complication, and by other forms of endemic disease, is still being noticed and discussed. But the *system* continues.

13. *With regard to New Arrivals,* the observation occurs, that recent arrival in India and youth are usually coincident in the same individual; they thus exert their double influence as predisposing causes of climatorial disease; to them also must be added inexperience and indiscretion on the part of individuals, leading to acts of exposure to exciting causes of illness that are avoided by older residents. Many years age, the circumstance was noticed and dwelt upon by medical officers, that new arrivals in India from temperate climates manifested a peculiar liability to be attacked with fever; that all Europeans require to be seasoned; and, in fact, in the West Indies, and on the West Coast of Africa, the term "seasoning" was formerly, and may still be, applied to the first attack of this nature. It was considered that this process was necessary before individuals came to that state of health best suited to a tropical climate; it was added, "the full, warm, generous state of body which belong to a temperate climate are not suited to a tropical climate, and Europeans arriving in that state are always brought down, often through some severe illness; but whether they have illness or not, they always alter, and it is necessary they should alter, in order to give them a chance of preserving their health in that climate." Thus the natural inference is, that in the one set of cases the seasoning process takes place as a violent cataclysm; in the other gradually, and without such extreme systemic disturbance. In the case of soldiers, the former is indicated as being the process of more frequent occurrence. According to official reports for 1874, of sixty-nine fatal cases of enteric fever, forty-seven of its subjects had been less than ten months in India, and only six over four years. But by the same record it is shown that this rate of mortality is not limited to what is called enteric fever; that out of 453 deaths, 120 took place in men of less than one year's service in India; in fact, that the relation of this mortality coincided with that of fever generally.

14. *Views of Medical Officers.*—Various comments were made by medical officers in regard to the conclusions expressed in the first special report now on the table. A summary of at least some of the views expressed in those comments indicates the following results, namely: 1. That "typhoid fever" is said to be "a perfect misnomer". 2. The theory of dirty localities is good, but the writer does not agree with this as an originator, though that dirt will develop any disease is undoubted. 3. There is evidently great obscurity as to the nature of typhoid fever, for we see

diseases of a totally different character, cholera, for instance, attributed to the same cause. 4. In many cases the simple continued fever seems to merge into the remittent, and *vice versâ*. 5. There is no doubt that malaria has done its share in producing fever. 6. That all fevers with infiltration of mesenteric and Peyer's glands, with elimination by ulceration or otherwise, as evidenced during life by diarrhœa, character of the stools, tympanitis, gurgling, high temperature, more or less stupor or delirium, are decidedly enteric, and that the cases called intermittent, remittent, and continued have been improperly classed. 7. That the writer does not mean to say the fever is not considerably, often it may be entirely, produced by climatic or malarial causes, but that the disease is one and the same as enteric at home he firmly believes. 8. It is thought to be an open question if it should not be called by some such name as tropical enteric in place of simple enteric. 9. That the circumstance of a patient convalescent from fever losing the hair off his scalp indicated that the nature of the fever from which he had suffered was enteric. 10. That the older writers on the disease wrote at a time when the identity of enteric fever was unknown, and that, therefore, it is not unreasonable to suppose that had they been acquainted with what has since been established, they would have considerably modified, if not altogether altered their opinion as to the etiology of this disease. 11. The poison, the manifestations of which present that group of symptoms called enteric fever, and which in fatal cases is invariably found associated with lesions in Peyer's patches and mesenteric glands, is as distinct and separate as any of the other animal poisons which produce specific results, such as small-pox, measles, scarlet fever, etc. 12. That wherever you find after death ulceration of the agminated glands of the lower third of the ileum, the disease is almost always one of enteric fever, whether the cause has been ascertained or not.

Thus, the inference appears to be in accordance with the summary given, that with regard to the several points referred to by medical officers, very different, if, indeed, not opposing opinions are entertained among themselves; also that differences of opinion exist with regard to causation, phenomena and pathological conditions in connection with what is termed "enteric" fever; while in regard to not one of the points enumerated is there concurrence of opinion.

15. *Conclusions.*—Finally, in a review of what has now been adduced, the following conclusions appear justifiable, namely: 1. That a form of fever attributed to endemic

causes, presenting similar phenomena to that now called synonymously typhoid or enteric, was described by ancient medical writers of India. 2. That more recently, the older medical writers described similar lesions to those now stated to constitute a specific form of fever, the result of filth received into the system, as being complications of endemic and climatorial fever; also, that the expression typhoid was used by them as indicating a particular *condition* of the patient, occurring in the course of fever, whether intermittent, remittent, or continued in type; not a form of fever specific in itself, and due to specific causes. 3. That the experience of British troops in India does not support the conclusion that the form of fever to which in recent times the term enteric has been applied, is dependent for its propagation on a poison of a specific fæcal character. 4. That a form of fever, like that which in Europe is called typhoid, except in its etiology, exists in India and other hot countries; that in them such a fever is due to climatic influences, not to filth or other specific causes; that all attempts made to trace any of 175 cases recorded to such causes, have been unsuccessfully made. 5. Various standards are adopted of what, by different individuals, is held to constitute the disease named by them typhoid or enteric fever. 6. The use, synonymously, of the terms typhoid and enteric, leads to the result that two different sets of conditions are thus brought under one heading, whether they happen or not to be united in the same case. 7. Neither of the recognised causes of specific enteric fever which are enumerated invariably give rise to that disease, nor is enteric fever the only form of disease that is induced by those causes, when they do operate. 8. Similar phenomena to those that arise from a specific cause are acknowledged to arise also as a result of fatigue, exposure, cold after exertion, etc. 9. But even in England all medical men are not yet agreed as to the cause of enteric fever. 10. A recognized authority on the subject makes this statement: "I do not believe that there is any necessary connection between filth and enteric fever, and, indeed, I doubt if there be any at all." 11. In Madras, pythogenic causes are, for the most part, non-existent, and where present, are inoperative, at least in so far as the cases recorded in the reports before us are concerned. 12. That in no instance among those recorded was an attack of so-called typhoid or enteric fever traced, by reporting medical officers, to the operation of a specific cause, either in or around their barracks, or in the bazaars frequented

by the men. 13. The phenomena, considered by certain writers to be distinctive of typhoid or enteric fever, have not been present in their entirety in the cases said to be of that disease. Some, or all, such phenomena occur in the course of other diseases, both in Britain and in India. 14. In three so-called typical charts, severally extracted from the works of recognised authorities on enteric fever, the line markings differ from each other absolutely in their character; similar differences occur also in the temperature charts attached to reports of particular cases described as of that disease. 15. In the cases recorded of so-called enteric fever in Madras, different kinds of eruptions occurred; but their relation to disease of Peyer's glands was not demonstrated by statistics. Also, eruptions precisely similar in nature occurred in other forms of disease, and in the absence of any apparent disease whatever. 16. The period of duration varies greatly in cases of specific enteric fever. Similar and equally great variations are observable in cases of endemic and climatorial, but otherwise non-specific, fever in India. 17. Specific pathological conditions of Peyer's glands and of the mesenteric glands are described as constituting the distinctive character or *essence* of enteric fever. According to other authors, conditions undistinguishable from those found in the bodies of persons who have died by specific typhoid or enteric fever occur also in those who have died by other diseases and by certain poisons; also, that cases of *typhoid* or enteric fever are recorded as occurring in persons at an age beyond that in which Peyer's glands are present. 18. Specific enteric fever in Britain prevails chiefly in autumn—that is, during the season when diarrhœa is most prevalent, and in which what was formerly called autumnal fever was chiefly observed. In India, ardent fevers chiefly prevail in the dry hot season; those with intestinal complications during the hot and moist period of the year. 19. The fauna and flora of particular districts are admittedly modified by climatorial and other influences affecting the locality. As pathological conditions, infraction of Peyer's patches is considered to be endemic in India. 20. The expression "malaria" is in these reports employed as synonymous with endemic and climatorial influences. 21. Imported animals into India suffer equally with if indeed not to a greater degree from those influences than does man. 22. In Britain, cases of "enteric" fever occur chiefly during youth and early manhood. In India, the relation of endemic disease generally to age corresponds pretty closely to that of "typhoid" fever in Europe. 23. New arrivals in India are specially liable to

be attacked by what has been termed "enteric" fever. But so also are they in regard to other forms of fever, and also by other endemic forms of disease. 24. According to the *résumé* of views already given from reports by medical officers, the opinions severally expressed by them are in absolute disagreement as regards every point touched upon having reference to what is termed "enteric" or typhoid fever in India, including definition, causation, phenomena, and pathological conditions. 25. But *if* in Britain such phenomena and pathological conditions are due to the operation of filth causes on the system, and *to none* other, the evidence adduced is amply sufficient to justify the conclusion that precisely similar phenomena and pathological conditions occur in India as results of endemic and climatorial influences operating on particular states of the system in individuals; and that those influences are of themselves alone amply sufficient for their causation.

Such, then, is the summary of views it has been my privilege to lay before you; such the conclusions I have drawn from them. If, in the comments that may be made on these remarks, any points still obscure shall have light thrown upon them, I trust that the paper now read may result in good, not only with regard to the purely professional points noticed in it, but also with regard to the well-being of our soldiers in India.

NOTES ON THE RELAPSING FEVER OF INDIA.

By NORMAN CHEVERS, M.D.

[*Read: January 7th*, 1880.]

The question—Is relapsing fever a new disease in India? may, perhaps, be still open to controversy, but I think that we have strong evidence upon which to base the conclusion that it is one of the indigenous pests of the country, and that its history may be traced back at least to the middle of last century.

Relapsing fever having been clearly recognised in Europe, especially by Drs. Henderson and Wardell, as a disease altogether distinct from typhus, in 1842, when it became generally prevalent throughout the United Kingdom, was first distinctly observed and described in India by Dr. Robert Lyall in 1852-53.

I have given a brief abstract of Dr. Lyall's report of the fever which carried off large numbers of the inhabitants of the Yusufzaie Valley, situated about forty miles from Peshawar, in my chapter on typhus, in the *Medical Times and Gazette*, page 120, vol. ii of 1879. The following were the chief characteristics of this disease:—It was epidemic, it was distinctly contagious, it was extremely prevalent, out of two hundred and twenty-six houses in one village, one hundred and thirty-one were found to have been visited by the disease; and of the nine hundred and forty-nine inmates of these houses, four hundred and eleven (or nearly one-half) were attacked by it. This fever presented a characteristic in which the relapsing fever of India has generally been found to differ from that of Europe—it was very destructive to life,—out of four hundred and ten attacked, one hundred and twenty-five had died, and one hundred and twenty-six remained sick when the report was made. At the commencement of the epidemic, jaundice was not an invariable complication; as the disease increased in prevalence, the body, in almost every instance, became jaundiced at about the fourth day, and a tendency to diarrhœa showed itself. Dr. Lyall wrote—"The most remarkable feature in this complaint is its tendency to relapse from two to ten days after the subsidence of the previous attack, without any irregularity on the part of the patient, in the way of exposure or diet, to account for it. These relapses are always attended with

great derangement of the bowels, and often seem to depend upon a low degree of gastro-enteritis. A large number of people are carried off from the chronic dysentery arising from this cause. The convalescence in all is remarkably slow, and many months are always necessary before their health becomes anything like re-established."

There were inflammation and suppuration of the glands in the groin, axillæ, and neck in some who survived the relapses. At the onset of the fever, quinine was not successful, but it was useful in the stage of relapse. The poor, uncleanly, ill-fed, and badly clothed and lodged were the chief sufferers. Thus, in the fever of the Yusufzaie Valley, we have displayed all the most distinctive characteristics of the relapsing fever of India—an extremely prevalent and destructive epidemic or endemic fever, arising among the poor and starved, and perhaps spreading to the well-to-do inhabitants by force of its contagiousness. The tendency to relapse is very marked, the convalescence is remarkably slow, there is generally jaundice, and quinine has no specific power as an antidote to the fever poison.

The fever observed by Dr. Lyall was clearly a disease very nearly allied to the Ictero-Hepatic form of relapsing fever, the bilious typhoid of Lebert, probably modified, as the continued fevers of India generally are, by the influence of marsh miasm. Thus it was noticed, in this and in other similar outbreaks, that "the relapses of the fever assumed the remittent or even intermittent type."*

India was long supposed to be as free from relapsing fever as from true typhus. In the edition of his treatise on *Continued Fevers of* 1862, page 301, Murchison says—"In India, and indeed in all tropical countries, relapsing fever is unknown." Other leading authorities maintained the same opinion. But in his edition of 1873, page 328, Murchison fully admits the occurrence of this disease in Upper India.

We have strong grounds for believing that the disease which Dr. Lyall first recognised in 1852 as relapsing fever was then by no means a malady new to India.

In his report on cholera, Mr. Jamieson has given a vivid narrative of an outbreak which ravaged Upper India in 1816. Throughout the Upper Provinces there prevailed most extraordinary drought and heat—such as had not been

* *Vide* Dr. Lyall's and Dr. Farquhar's reports on this fever—*Indian Annals of Medical Science*, No. 3, p. 16, and No. 4, p. 504. It will be well that readers who are not experienced in the diseases of India should, as far as possible, consult and study the originals of the many valuable reports to which I shall have to refer, and of which the scope of my notes permits me only to give brief abstracts.

experienced since 1803. To the long-continued drought succeeded heavy and incessant rain, so that the whole face of the country was laid under water. The disease appeared among the natives in August, and raged epidemically in every city and town between Patna and Seharunpore. "It exhibited itself in the shape of a bilious remittent fever of a violent inflammatory type, accompanied, like the yellow fever of the West Indies, with suffusion of the skin." Its tendency was to terminate fatally at the end of two or three days. "The width of its range precluded the possibility of its being referred to any causes purely local." Its attacks were indiscriminate; "it seized equally Europeans and natives, and as readily entered the open and spacious house of the officer and civil servant as the crowded barrack of the soldier" [barrack accommodation in India was then generally very faulty] "and the low filthy hovel of the native." The mortality in the largest cities was very great. "Of numerous native villages nearly the whole population was ill at one and the same moment, and many of the shops were shut for want of people to attend them. The banks of the river were covered with the dead and the dying." "In Delhi, of two native corps alone, there were five hundred men at once sick in hospital." The European troops suffered quite as severely. "Of the European flank battalion, six hundred and forty-eight strong, stationed at Allahabad, three hundred and five men were attacked during October; early in November there were, at one time, one hundred and eighty rank and file, and twenty women and children on the sick list; and, in the whole, only seventy men of the seven companies of which the corps consisted escaped the disease. But Cawnpore was the principal seat of its ravages amongst Europeans: of four King's corps, the 24th Regiment L.D., seven hundred and thirty-five strong, and the 14th, 66th, 87th Regiments of Foot, eight hundred and fifty-two, eight hundred and thirteen, and seven hundred and two strong, then in cantonments, there were, from first to last, nearly one thousand persons taken ill. The disease here began in August, prevailed during the three succeeding months, and did not finally withdraw until the setting in of the cold weather in December. It was at its height in September and October; eight or ten, or sometimes even fifteen men then died daily. The 87th and 66th Regiments suffered dreadfully; from the time of their reaching the station they lost nearly four hundred men. The former corps is said to have had one hundred and nineteen in hospital at once, to have buried twenty-one persons (including women and children) in one day, and

upwards of ninety of its number within a month." "This", adds Dr. Jamieson, "is a degree of mortality far exceeding anything then on record in the medical annals of Bengal."

Considering that this report was written twenty-six years before relapsing fever was recognised in Europe, and remembering that other Indian fevers are not characterised by a tendency to relapse, the following statement made by Mr. Jamieson must, in connection with the rest of its history, be taken as clearly showing that this malady was Indian relapsing fever. "It has been stated that the disease abated in December" [it commenced in August]; "so early as the end of November its attacks had become less frequent, and its symptoms milder; *but relapses were still common*,* and it was very difficult to counteract the extreme debility invariably consequent on the attack."

It is particularly noteworthy that the first visitation of the Pali Plague prevailed in Western India from 1815 to 1819, and that the outbreak of relapsing fever described by Jamieson occurred in the Upper Provinces only a few months previous to the commencement in Bengal of the great cholera epidemic of 1817. So also when, in 1836, the Pali Plague, commencing in Marwar, spread rapidly and attacked thirty-two villages of Mewar, a destructive fever, which must have been relapsing fever, made its appearance in the town of Bareilly,† and committed serious ravages in Rohilkund, in Mynpoori, and in the districts around Delhi. In tracing the history of relapsing fever in India, it is interesting to observe that, in his narrative of the outbreak of this disease at Bareilly, Mr. Guthrie states that "It is reported that about the year 1818 [when, it must be repeated, Pali plague prevailed in Western India], during the medical charge of Mr. Evans, a similar fever prevailed, sacrificing three hundred victims; reappearing under Mr. Browne in 1818, and causing great mortality from July to September; and that it also paid a passing visit, under Mr. Rhodes, in 1834, of only a few days, destroying eighteen convicts." The reports show clearly that this disease was contagious, but not so extremely contagious as the fever of 1816. Mr. Guthrie says on this point, "As to its infectious character here, within certain limits, I have no doubt but it has laws of its own. When the town was

* The italics are mine.—N.C.

† The history of this disease will be found in the following reports:—Hugh Guthrie (Bareilly), *Transactions of the Medical and Physical Society of Calcutta*, vol. viii, part II, p. 189; and of Dr. D. Macnab (Mainpuri), *ibid.*, p. 230; and those of Stuart, Spencer (Moradabad), and Shirreff (Delhi Territory), in Dr. James Rankin's "Report on the Malignant Fever called the Pali Plague," p. 196 *et seq.*

devastated by it, the convicts had it not among them, but now it is *vice versâ*." Jaundice usually characterised this fever, the eyes being of a deep saffron colour. The disease was not, at its outset, amenable to quinine; severe pains in the bones were grievously complained of; there was great tendency to bowel irritation; recovery was extremely slow. The malady prevailed almost exclusively among the poor and among prisoners crowded into ill-constructed and deficiently provided jails. Dr. Stewart, of Moradabad, wrote, "Persons living in well-ventilated places entirely escaped this epidemic", and "persons who could afford themselves good food, even though living in indifferently ventilated situations, provided they avoided exposure to the infection, also escaped." Most of the medical officers reported very unreservedly upon "the ill-housed, overcrowded, badly clothed, and ill-fed condition of the prisoners". The destruction of life was very great. Mr. Shirreff reported that, at one village in the Panipat district, nearly two hundred persons died, out of a population of five hundred families, within six weeks; at another place, two hundred and thirty were said to have died within twenty-seven days out of a population of some one thousand inhabitants. In one division of this village, almost everyone who was attacked died. Between October and February, inclusive, the monthly range of deaths was from nineteen to thirty-two out of a body of sick averaging from ninety to two hundred and forty-five. Considering that, at that time, relapsing fever had not been differentiated, the evidence that this was the relapsing fever of India is very strong. Shirreff noticed that, in the Panipat and Rhotuck districts, "in the majority of cases the fever runs its course till the 7th, 8th, or 9th day". Macnab says that, at Mainpuri, "when the disorder proved fatal, the issue did not long remain doubtful; the course towards this consummation was run over rapidly, and five or six days may be given as the maximum period". Some were dealt with much more summarily. Spencer says, in describing the disease at Moradabad, "The patient died sometimes on the 3rd, but more commonly on the 4th to the 6th day". It is to be borne in mind that, unlike the allied disease in Europe, the relapsing fever of India is of such extreme deadliness that it frequently destroys the patient so early that the tendency to relapse cannot be observed in many cases. Thus the reports show that many died early in the disease from head, chest, and abdominal complications; and Guthrie wrote, "Many die in the first paroxysm, completely overpowered, as if the powers both of body and

mind were at once blighted. This tendency to sudden death, from shock, immediately the malady, in its overwhelming power, strikes the patient down, has been observed in plague, in cholera, and in variola." Spencer, describing the Moradabad fever, makes a statement which, standing alone, might be taken as sufficient evidence that this malady was relapsing fever: he says, "Many men suffered severe relapses which mostly terminated fatally": but, while I consider that our mass of evidence in favour of this view is quite weighty enough to enable us to dispense with this striking statement, it is, I think, clear from the context, that the writer used the expression "relapses" as synonymous with "sequelæ", which were here dysentery and large abscesses. But the evidence of Guthrie and Macnab on this point is very strong and direct. Mr. Guthrie had a convalescent hospital where those discharged from his fever hospital were accommodated and placed under the care of an assistant, who was ordered "*instantly as relapses occurred to send them back to the hospital bungalow;* from these again, as they recovered a little strength and power of digestion, a selection was daily made, and placed in another outhouse under the care of the native doctor of the Insane Hospital, who saw them fed, exercised, etc., and, if these *did not in five or six days suffer a relapse,* they were sent to the jail as cured. The grand difficulty was to restrain them from eating, as the indulgence in even a moderate quantity of solid food *re-induced the fever* or caused fluxes."*

Macnab wrote—"Low delirium was very common, and when a torpor supervened, the case might be considered hopeless. We at times, however, had cases where the stupor partially vanished, and the intellect became in some degree clear; but this gleam was evanescent, for suddenly a relapse would dissipate the illusive hope, and the patient fall back into a state worse than that from which he had partially emerged. If, during the first day or two of improvement, the slightest retrograde movement were made, hardly an individual recovered."

The above facts convey a very important lesson to the young practitioner. He must not resolve to deny that a fever which suddenly appears among the poor of the town, or the prisoners in his jail, is Indian relapsing fever—solely upon the ground that none of his earlier cases are marked by anything like the cessation of fever followed by a distinct relapse of fever. If the type is nearly that of an ex-

* The italics are mine.—N. C.

tremely severe remittent, with or without jaundice, and not amenable to quinine, and if there be substantial evidence of contagion, and the spirillum be detected in the blood, the disease is relapsing fever. Still it is clear that, whenever an outbreak of Indian relapsing fever is carefully observed throughout its course, positive evidence of existence of the characteristic tendency to relapse will not fail to present itself.

As I have already mentioned, Dr. Joseph Ewart's experience when attached to the Meywar Bheel Corps, after he had observed cases of typhus and true enteric fever in the Ajmeer jail in 1855, led him to inquire, in the *Indian Lancet*, whether many of the so-called malarious remittents are not, in reality, typhus, typhoid, and relapsing fever.

The last commentary on Indian fevers published by Dr. Kenneth Mackinnon,* was a note on "Fevers with yellowness of skin", published in the *Indian Annals of Medical Science*, No. 7, 1857, page 155. It may be taken as a useful lesson to many of us to observe how—in reviewing, for a second time, Jamieson's narrative of the epidemic fever of 1816, and the accounts of the pestilential fever of 1836-37, and comparing this disease with the Yusufzaie fever; and while manifestly yielding to the force of evidence towards the conclusion that these were outbreaks of relapsing fever, and frankly admitting that they could, at most, have been, if of miasmatic origin at all, only in part due to paludal influence—this admirable observer and absolutely truthful reasoner still clings to his old traditions, arguing that marsh poison acted here in combination with animal and vegetable filth; that Dr. Maxwell appeared not to consider the fever he saw at Dera Ghazee Khan contagious; that yellowness of the skin, glandular swellings, and hæmorrhage, which attended these fevers, assimilate them more to the plague than to any other disease; and adroitly urging that this deadly Indian fever can scarcely be identical with the by no means destructive relapsing fever of Europe described by Jenner. A careful perusal of these four most interesting pages will, however, show that his opinions had begun to change, and that, in all probability he would have been among the first to recognise the true nature of the relapsing fever which made its appearance in the jails of the North-Western Provinces in 1860.

Several years, however, had still to elapse before the occurrence of relapsing fever in the jails of Upper India was

* I was wrong in saying at p. 363, vol ii, 1879, of the *Medical Times and Gazette*, that the last of Dr. Mackinnon's writings on Indian Fevers was published in 1855.

generally suspected. A very destructive contagious fever appeared in the central jail at Agra, in 1860, and was described by Dr. Walker. It again prevailed, in a very deadly form, in the Agra jail in 1864; and, in that and the following year, it appeared in the jails of Umballah, Futtehghur, Allyghur, Jyepore, Malwa, etc. It was described by the several reporters as true typhus, contagious fever, typhoid fever, and, in the words of the Sanitary Report—"Some again believe it to be identical with the relapsing fever of Europe." Dr. Gray, Dr. Beatson, and Dr. Dallas clearly recognised this disease, as it occurred at Agra, Lahore, and Umballa, as relapsing fever. The whole question was most fully and ably reviewed by Dr. David Boyes Smith, in the *Indian Medical Gazette* for 1867 and 1868; and, although his verdict met with a good deal of opposition (especially from the Medical side of the Indian Sanitary Commission) he, I consider, brought forward very strong arguments in favour of the opinion that the contagious fever of the up-country jails was nearly identical with the relapsing fever of Ireland, Scotland, Germany, and America. Indeed the Sanitary Commission went so far as to admit that "the disease resembles the famine fever of Europe in many important particulars."

As we have already seen, the relapsing fever of India is distinguished from the European disease in being attended with great mortality. The fever at Agra killed 406 prisoners in 1860, and 250 in 1864. The opponents of the view held by Lyall, Ewart, Gray, Bateson, and Smith insist strongly upon this point, and also deny that famine had anything to do with the causation of this disease in the Indian jails. These arguments are met by the facts that, although it is not a matter of necessity that an Indian famine should be attended with relapsing fever—the last famine of 1876-77 in the Madras Presidency had not this aggravation—the relapsing fever of India always originates either among the poor or the prisoners, and that, at least in the Punjab, the Lieutenant-Governor, Dr. Dallas, the Inspector-General of jails, and Dr. Gray, so fully concurred in the belief that the appearance of this fever was clear evidence of a bad hygienic condition of the jails, that orders were given making great improvement in the diet, lodging, and clothing of the prisoners.

It can hardly be expected that men who examine this fever from a scientific point of view, by the light of Indian experience, will be shaken by the personal and political arguments that this is not relapsing fever because it devas-

tates certain districts in which famine has not recently occurred, or destroys 25 per cent. of the prisoners in several central jails under the eye of a humane government and of officers whose administration has been reduced to a system which is nearly perfect. I have already argued (page 395 of vol. i, *Medical Times and Gazette* for 1879) that the poor of India are the descendants of many generations of starvelings, and that they are, by reason of the deficiency of nutritive elements in their food, subject to the life-long misery of chronic famine. Of these poor the majority of the jail population are the most singularly unhappy representatives. The axiom, generally applied to the European criminal, that he represents the worst blood, the largest sum of constitutional disease, and the lowest feeding, clothing, and lodging discoverable in his city, is equally true of the ordinary Indian prisoner whom destitution frequently precipitates into crime. Again, although it may have long been true that the food allowed in an Indian jail is somewhat better and more ample than that obtainable by the very poorest free inhabitants of the district, its power of maintaining health under the amount of labour exacted is not always unquestionable. There still, again, remains the very serious question—Do the prisoners actually obtain the authorised allowance? In reply to this I may give one or two facts which were painfully forced upon my notice during twenty-six months in which I officiated as Inspector-General of the fifty-two jails in Bengal. I may premise that, at my first large civil station, Chittagong, I once arrested the attendants as they were carrying out of the front door of the jail, in broad daylight, close past the lodge where the darogah (native jailor) usually sat, a huge basket of hot rice. When I reported this occurrence to the magistrate he appears to have held some inquiry, the result of which was that it was intimated to me that he had ascertained that the prisoners were accustomed to send what remained from their meals for the support of a favourite Brahmany bull which infested the neighbourhood of the jail. To this I felt constrained to reply that the quantity of rice which I had seen was, self-evidently, in excess of the gastric capacity of any Brahmany bull, however voracious; that the grain was not leavings, but perfectly white rice fresh from the cooking vessels; and, further, that my acquaintance with the physiology of the Brahmany bull taught me that, however capable that sacred animal might be of eating an immoderate allowance of simple rice, it was difficult to see to what dietetic use, he being a strict vegetarian and quite above all suspicion of addiction to stimulants, would apply that large

allowance of *marsalla*—composed of dhall, oil, strong spices, and capsicum—which I saw lying upon the heap of snowy rice. Further, that a very large majority of the prisoners were Mussulmans, who care nothing whatever for the Brahmany bull, save when he appears before them in the form of appetising *kiwabs*. Many years afterwards, when I held temporary charge of the prisons, the superintendent of the Alipore jail reported that a large batch of convicts had arrived from Midnapore in a starving condition. I immediately wrote to Midnapore, which is about a night's post-journey from Calcutta. On its way, my letter was crossed by one from the very able joint magistrate, informing me that his jailor—one of our pattern jailors—had been detected in robbing the prisoners daily of large basketfuls of cooked rice. Soon after this I sent a circular to magistrates, requesting that, at a certain hour on a certain day, they would test all weights used in weighing food in the fifty-two jails of Bengal, by having them weighed against rupees in their presence. The result was that scarcely a single weight came up to the just standard, the weights of the pattern jails being generally most deficient: "The scales were out of gear," "The weights were worn," etc., etc.! Until you can fill your jails with perfectly healthy, well-nourished felons, and can provide that they shall not be over-tasked and under-fed, you may not in reason presume to maintain that, when a contagious fever appears and kills off 25 per cent. of your prisoners, it cannot possibly be of the nature of relapsing or famine fever.

To continue the history of relapsing fever. In April 1877, Mr. Blayney, a medical practitioner and a member of the Town Council of Bombay, stated that a fever which had appeared among those lodged in the relief camp on the Flats was not the ordinary malarious (marsh remittent) fever, but was "a contagious epidemic disease—a relapsing fever". The Government immediately desired Surgeon-General Hunter, the head of the Medical Department, to institute inquiry into this fever. Dr. Hunter appointed Dr. Hewlett, the Acting Sanitary Commissioner, and Dr. H. Vandyke Carter to this duty. The report of the Committee and Dr. Hunter's report appeared in the *Indian Medical Gazette* for October 1, 1877. The Committee's report appears to have been somewhat premature and to have been founded upon insufficient data and observation, but it contained the important announcement that Dr. Carter had discovered in the blood of these fever patients the *spirillum* already recognised as characteristic of the relapsing fever of Europe. Dr.

Hunter's opinion was opposed to that of the Committee, he maintaining that evidence was wanting to prove that the disease was relapsing fever. An important report of the whole investigation has been given by Dr. H. Vandyke Carter.* Dr. Carter's report is so recent and may be so readily consulted that it will be sufficient to cite a few of its leading statements. He shows that crowds of indigent peasantry, flocking as refugees to Bombay, brought with them not only destitution and overcrowding, but also a contagious fever, which soon spread to the resident population immediately in contact with them, and became the cause of death to many. He regards the disease as famine or relapsing fever. The report is founded upon the examination of about 350 cases of this fever. Quinine had no specific action in this disease. Both at the height and towards the close of the epidemic, Dr. Carter says cases occurred which had all the characters of the so-called "typhus icterodes" or "bilious typhus", in a most pronounced degree. In these cases there was generally "a yellow tinge in the conjunctivæ". "Jaundice was commoner here than it usually is in" [the relapsing fever of] "Europe." When at its height, about the seventh or eighth day, the general distress abruptly came to an end with critical sweating and considerable fall of temperature. There occurred from one to four relapses in 50 per cent. of the cases. Abortion in pregnant women occurred in the Bombay fever just as it does in that of Europe. The ravenous appetite, so generally observed in this type of fever, was often noted. The average death-rate was about 10 per cent. of those attacked. A marked characteristic of this fever was the presence of the *spirillum* (Spirochæte Ehr.) in the blood, in immense numbers, "often incalculable except by hundreds of millions," in some cases, rarely in others. They were often so crowded as to offer mechanical impediment to the circulation, evidenced by dusky lividity of the countenance. The parasite was sought for in many cases of remittent fever and ague, but was not found. It was not found in enteric fever (except in one very doubtful instance) or in small-pox, varicella, or symptomatic fevers. Dr. Carter says, "Once indeed I found a minute and active organism in the blood of a patient affected with fever, seemingly intermittent in type; but, in general, I conclude that the spirillum must be regarded as distinctive of one clinical pyrexial form." In the *Lancet* for June 7, 1879, page 814, Dr. Carter announced that the spirillum

* *Medico-Chirurgical Transactions*, vol. lxi, p. 274, "Notes on the Spirillum Fever of Bombay". 1877.

fever may readily be communicated to the *macacus radiatus*, a common quadrumanous inhabitant of the Bombay forests, by inoculation with the blood of a fever patient, or with that of an infected monkey, with the result of marked general symptoms, and abundance of the attendant blood-parasite. *Post-mortem* appearances, too, are equally accordant.

As I have already shown, Dr. Lyons, one of the most laborious modern investigators of Indian fevers, Mr. Oldham, and others, absolutely deny the existence of paludal poison. Mr. Oldham, however, believes in the separate existence of that Indian remittent fever which most of us characterise as paludal. Dr. Ewart long ago contended that some cases of so-called paludal remittent would, upon closer investigation, be found to be true enteric, typhus, or relapsing fever, complicated with, and masked by, the marsh poison. Dr. Lyons has published two elaborate works* in exposition of his doctrine that Twining, Morehead, and many others of us have been deceived in believing that there is, or ever has been, such an entity as paludal remittent fever in India, but that all such cases, carefully observed, will prove to be examples of continued fever—enteric, relapsing, or typhus. Many of Dr. Lyons's contemporaries will live and die in the conviction that paludal remittent, curable by quinine, is prevalent all over India (otherwise why not do away with quinine?); but we must all feel grateful to Dr. Ewart and to him for the manner in which they have illustrated the necessity of sifting our cases of grave Indian fevers, and of eliminating those of true enteric and of relapsing fever, all of which were, but a few years ago, either entirely confounded with, or, according to the sagacity of the practitioner, more or less imperfectly separated from the "remittents", and were invariably reported and embodied as remittents in the returns. A careful perusal of the third section of Lyons's Treatise will lead to the conclusion that relapsing fever has been far more prevalent in the East than was generally believed. Of this we have many illustrative facts before us. To give one example out of a score:—Dr. John Macpherson has recently informed me that, during the Burmese War in 1852-53, he received communications from Dr. Murphy, who had served in Africa and in the West Indies, insisting that a disease which prevailed in the European force in Burmah was perfectly identical with true yellow fever. The reports of the 80th and the 18th Royal Irish Regiments, by Messrs.

* "Typhoid Fever Discriminated from Remittent Fever," etc. (*Indian Annals of Medical Science*, No. xxviii, 1871, p. 241), and "A Treatise on Relapsing or Famine Fever". London, 1872.

Taylor and Stewart,* show that, in the first of these regiments, the deaths were 19 to 796 admissions for fever, and that in the second corps they were also 19 to 796 admissions for fever. The icteroid character was well marked. Contagion does not appear to have been suspected.

It was, some years ago, well remarked that, whenever a fever case in India puts on an icteritious appearance, especially in a jail, we ought to be on our guard against an outbreak of relapsing fever. Every such case should be thoroughly sifted and closely watched. True jaundice is not, and has not been, as far as I am aware, a characteristic of the paludal remittent of any Indian locality. It is, of course, needful to distinguish the pallid, dusky greenish or yellowish cachæmic aspect presented by many sufferers from the worst paludal fevers from that seen in true icteroid fever. Some years ago, I treated several of a party of European workmen, most of whom were attacked with serious remittent fever while setting up an iron bridge at Peealee, on the Mutlah Railway, about thirty miles from Calcutta. The aspect of these patients was so characteristic that I, more than once, on entering the ward and glancing along the beds, said, "That is a fresh case of Peealee Fever!" This, however, was not jaundice; it appeared to be due to disintegrative blood change.

The question—*Does relapsing fever occur in the Lower Provinces of Bengal and in Calcutta?* is one which still requires careful investigation, even by those of us who have practised nearly all our lives in that part of the country. There is no positive evidence to show that the disease has appeared, within this century, in any town or jail of the Lower Provinces. The result of my own experience of upwards of twenty-seven years in malarious places is that, when I review the subject, my mind reverts to the case of *one* native who was brought into my ward moribund in fever, with saffron-coloured eyes and sordes about the mouth. Kenneth Mackinnon repeatedly insists that he could never trace any evidences of contagiousness in remittent fevers prevalent in and below the Province of Behar. Dr. Francis Anderson's committee, who carefully investigated the Burdwan fever in 1864, were convinced that it was not contagious; and this opinion has, I believe, been held by all who have since observed this disease, with the exception of Dr. Lyons. I, however, think that few Calcutta medical men will be able to concur with him in thinking that Burdwan fever, which he terms "the non-eruptive disease of Bengal", "communi-

* *Indian Annals of Medical Science*, No. ii, 1854, pp. 418 and 431.

cated the eruptive and milder variety" [dengue] "to the residents of the wealthy and well-cared for metropolis".* My friend Dr. William Palmer has lately informed me that he had large opportunities of studying relapsing fever in England; but that, during his long Indian career, a great part of which has been spent in the Calcutta hospitals, he has never seen a case of this disease. Still the following points appear to claim attention. The fevers with which Calcutta was visited between the time of its first occupation, late in the seventeenth up to the beginning of the present century, were almost unaccountably deadly. We read of 460 burials out of 1200 British inhabitants of Calcutta between August and the ensuing January,† and of a detachment of 240 men, chiefly Europeans, stationed at Fultah, on the Hooghly, of whom not thirty were left alive between August and December.‡ Dr. James Lind wrote of the fever which prevailed in Bengal in the year 1762, and which he terms "putrid and remitting marsh fever", as "raging more or less in different places according as the soil was more or less swampy", and as becoming so violent, especially during the rainy season, as to end in death during the third fit, which was generally the case, while others were exposed to the danger of dying at every fit. Admiral Stavorinus, speaking of the fever which prevailed among the European inhabitants of Calcutta between 1766-71, says that "it generally sweeps away those who are attacked by it in the course of three days". Dr. John Clark describes "fever and flux" as being very destructive in Calcutta between 1768 and 1771. He states that fever frequently carried off the patient in twelve hours.§ Assuredly these are not the characters or the death statistics of the paludal remittents of the present day! They more resemble those of the relapsing fever of 1816, in which it was calculated that, between the middle of January and the end of June, there were 8,500 deaths out of a population of about 55,000.‖ The evil sanitary condition of old Calcutta, the prevalence of sea-scurvy in ships' crews, and inappropriate treatment, prejudicial as they undoubtedly were, are inadequate to account for such death-rates as these. We have very little precise information with regard to the type of these Calcutta fevers; but Dr. Lyons notices that Dr. Lind wrote of

* Treatise, preface, p. vii.
† Captain Hamilton, between the years 1688 and 1723.
‡ Ives, in 1756.
§ Ranald Martin's *Diseases of Tropical Climates*, second edition, p. 308.
‖ Kenneth Mackinnon.

the deadly Bengal fever which he observed in 1762, that the "patients are very subject to relapses"; and also that, in Ives's narrative of the sickness of Admiral Watson, who died in Calcutta, on the third day of fever, in August 1756, (when the frightful mortality from fever alluded to above was going on in Major Kilpatrick's detachment down the river at Fultah), it is mentioned that "his eyes were a little yellow". Further, that, in the "Selections from the Records of the Calcutta Presidency General Hospital", published by Mr. Alexander Grant in No. ix of the *Indian Annals of Medical Science*, there are two fatal cases of fever occurring in 1797, in which it is noticed that, late in the disease, "the skin was tinged yellow" in one, and "the skin was considerably yellow" in the other case. I see that both of these patients died on the eighth or ninth day of the fever. Henry Goodeve notices* the striking fact that, in the year 1770, during a scarcity of rice, nearly 2,000 Europeans, besides 80,000 natives, died of fever in Bengal alone. All these points combine in countenancing, to say the least, a strong suspicion that relapsing or famine fever occurred in Lower Bengal until late in last century. With regard to more recent times, Twining says, in 1835:† "We every year meet with some patients in whom an intense yellow suffusion of the skin occurs in fevers of considerable severity." When treating of that very grave disease which he termed the "congestive fever of the cold season", he says,‡ "Intense yellowness of the skin and eyes, with red-coloured urine, are occasional attendants on these cases"; and further on, in speaking of this fever as it occurred in natives, he says, "Jaundice is by no means a rare sequel of the fever of this season". Dr. John Macpherson informs me that, in a pamphlet which he wrote in 1856, he remarked, "Cases do occur in which there is much yellow suffusion with a certain amount of black vomit, and such cases I have seen in Calcutta". In the preface to his *Treatise*, Dr. Lyons says, "Since the manuscript of this work was transmitted to England in October 1871, I have personally observed two epidemics of relapsing fever in Bengal". He appears to allude to the epidemic of dengue; but I think that we must await a fuller exposition of Dr. Lyons's views on this subject before we can attempt to argue the question further. I practised throughout the dengue epidemic of 1855, and during the first three months of that

* "Sketch of the Progress of European Medicine in the East", *Calcutta Med. and Phys. Trans.*, vol. viii, part II.

† *Op. cit.*, vol. ii, p. 201. ‡ *Ibid.*, p. 349.

of 1871; and, with a clear recollection of most of my numerous cases, I am unable to think that I witnessed anything at all resembling true relapsing fever in either of those epidemics.

Treatment.—1. *Prophylaxis.* Removal of troops from impested localities, careful segregation of sick, good food, clothing, ventilation, and general hygiene, quinine and iron in tonic doses. As few prisoners as possible to be kept in jails. A famine was threatened in Behar when I was officiating as Inspector-General of Jails. I recommended to Government that summary punishment should be employed in all minor offences, that the jails might not become overcrowded.

2. *Treatment of the Disease.* The principal indications are to support life beyond the eighth day, and to counteract the tendency to sudden collapse by liquid nourishment, wine, and quinine; and then to watch the patient vigilantly, sustaining his powers and averting all injurious and depressing influences, until danger of relapse is past, and true convalescence is well advanced.

3. To give quinine steadily in moderate doses, first as the TEST of the true nature of the fever; and next, when we are certain that we have relapsing fever to deal with, as our most powerful nervine tonic and as an antidote to whatever paludal influence may be present in the system. To watch for and meet cerebral, thoracic, and abdominal complications. To prevent or relieve bowel-irritation, to which there is generally a great tendency, the exciting cause being often improper solid food eaten to appease the great hunger which is usually present late in the attack.

4. To aid the always slow convalescence, by quinine, iron, and proper nourishment, and by change of air.

ON REMOVAL IN EPIDEMIC CHOLERA.

By Surgeon-General JOHN MURRAY, M.D.

[*Read: May 5th*, 1880.]

I am anxious to submit the following remarks on the subject of removal into tents in epidemic cholera, as it has not hitherto been discussed in the various papers that have been submitted to our observation.

The statistical papers I now desire to bring to your notice furnish valuable facts in the history of cholera in India since the beginning of the present century. They contain information which confutes some prevailing theories, and show that we can not only employ means to avert the disease, but that we can also employ means to remove it.

Cholera scarcely appeared in the medical returns, from whence these tables are drawn, up to the year 1817, when a sudden severe outbreak took place in Bengal, and rapidly spread all over India. The disease has continued to appear, at irregular intervals, up to the present time, in all the provinces. Returns have been regularly furnished to Government from the European troops, native troops, and native prisoners, and these furnish the basis of these tables. They commence in 1803 in Madras and Bombay, and in 1814 in Bengal. Cholera appears to have been entered under the head of "other diseases" previous to that date. The mortality shown varies considerably in the different Presidencies, particularly amongst the European troops and native prisoners; but it continued high until the year 1862, since which time there has been a marked diminution. To indicate the cause of this saving of life, and to show that it is contingent on the removal of troops and prisoners into tents, on the appearance of an epidemic, is the object of the present paper.

My experience of the benefit of removal commenced while I was in charge of the central prison at Agra, where I witnessed its good effects on the native prisoners. In 1851 there was a severe attack of cholera amongst them. It appeared on the 25th July, and the last case occurred on the 29th August, having lasted thirty-six days. On the

30th July, about one-half of the prisoners (1,400) were moved from the jail into the ancient buildings at Secundra, the Taj, and the Rambagh. From the 30th July there were 54 cases and 16 deaths amongst these; whilst amongst the 1,500 who remained in jail, there were 199 cases and 59 deaths. In 1856, there was another very severe attack in the same prison. Six cases of cholera were sent into the hospital from a local jail in the city on the 23rd and 26th May. The first case among the prisoners occurred on the 30th May, and the last case on the 20th July. There were 50 cases, and 21 deaths, up to the 12th June. Six hundred men were moved into the old buildings in the vicinity, on the 13th June, and 500 more into tents on the 28th June, and 700 more on the 3rd July. Out of a strength of 3,704, there were 349 cases in the jail, and 215 among those sent out, with a total mortality of 239.

In 1860, there was a very severe attack of epidemic cholera at Agra, in which the European troops suffered much, and I moved them into the old buildings, which had been occupied by the prisoners in the years 1851 and 1856, as well as into tents. This prevented the buildings being available for the prisoners, who were attacked with great intensity on the 8th August. In consequence of rain, they were not removed into tents until the 25th August. During these sixteen days there were 754 cases, and 158 deaths. On the 26th there were 30 cases, but in the following seven days there were only 38 cases, and only 3 more up to the 8th September, when the disease disappeared. Out of a strength of 2,063, there were 807 admissions, and 172 deaths. The delay in moving into tents was much to be regretted, as the danger from exposure, anticipated by the superintendent, was not shown to exist by the experience of the European troops who were moved into tents at that time during the rainy season. In 1861, another severe attack took place in the same central prison. It broke out on the 7th July, and lasted till the 10th August—thirty-five days. Eight hundred and fifty of the prisoners were moved into tents on the 10th July, after 50 cases had appeared, and there had been 16 deaths. There were only 4 cases in the camp. Out of a strength of 2,959, there were 173 cases, and 65 deaths.

In 1862, there was a mild attack in the Agra prison. The first case was on the 10th August, and the last on the 18th, a total of nine days. The prisoners were moved into tents on the 12th, and the disease subsided. There were, in all, out of a strength of 2,667, 23 cases and 12 deaths.

In 1856, at Agra, during the attack which I have described in the central prison, the cholera epidemic was very severe amongst the European troops in cantonments. I suggested to the Superintending Surgeon that they should be moved into tents; and, after a medical consultation, some of them were marched to the Rambagh. But the tents did not arrive till 12 or 1 o'clock, and the men were exposed to a bright August sun. The cholera subsided, but cases of sun-stroke were numerous; there were upwards of fifty during that and the following day. The men were, therefore, recalled to cantonments, and the measure of removal strongly condemned. The failure I attributed to mismanagement, in not having the tents ready to receive the men.

In 1860, I was deputy-inspector-general at Agra, and on the outbreak of cholera in cantonments amongst the European troops, I recommended the officer commanding the Brigade (General Showers, C.B.) to remove the regiment into camp. He pointed out the unfavourable result of the movement in 1856; but at the same time said, "it was a medical question, and that what I advised he would carry out; only, I must recollect the responsibility would rest entirely on me". This I accepted, and the troops were immediately moved into tents. The result was highly satisfactory, contrasting favourably with that of the movement in 1856, and also with the course of the disease at the same time in the Central Prison, before the prisoners were moved into tents, as previously detailed.

At this time the disease was very severe amongst the European troops at Muttra, and General Showers requested me to visit that station, which I did the next day. I found that the European artillery there had been decimated during the previous week—twelve out of fourteen cases had proved fatal, and another in hospital was dying. The medical officer said that medicine had no power to check the disease. I advised the troops being moved into tents, which was done that afternoon, and afterwards there was only one slight case, which recovered. It was on this occasion that I took a hint from a mistake which had occurred at Agra a few days before. A company of the 3rd Battalion Rifle Brigade had been sent into camp, and I had given a box of cholera pills to the serjeant-major who accompanied it, with instructions to give one to any man who went to the rear after gunfire. On enquiring next morning how many had been expended—"I used them all, Sir", said he, with a military salute. "All!" said I. "Yes,

sir, served them out to each man, with his grog." At this, there was a laugh at my expense! but the effect was so good (for there was not a case of cholera in the detachment afterwards) that I employed the same measure with the Muttra troop of Horse Artillery, and continued the plan in subsequent epidemics. It invariably secured the men a sound night's rest, and doubtless aided nature in eliminating the poison from those in whom it was present in a dormant state. During this attack, in 1860, the 3rd Battalion Rifle Brigade, out of a strength of 1,000 had 220 cases and 57 deaths. The first case was on the 9th August, the last on the 13th September, thirty-six days. They had improved at first, after removal, but there was a severe recurrence on the 30th August. The battery of artillery at Agra, at this time, out of a strength of 120, had 27 cases and 7 deaths.

In 1861, cholera appeared among the European troops at Agra, on the 18th July. It became severe on the 24th and 25th, there were 9 deaths on the 24th, 8 on the 25th. They would have moved into camp on the 26th, but rain poured down incessantly; during a break, on the morning of the 27th, Her Majesty's 42nd regiment moved from their barrack to the corridors round Akhbar's Tomb at Secundra. There were two fatal cases on the day they moved out, and one on the next day. On the 30th they went into tents. There were no more fatal cases. Out of a strength of 412, there were 47 cases and 24 deaths.

This epidemic was very severe in the cantonment of Gwalior (Morar). The first case appeared in Her Majesty's 27th regiment, on the 24th July, and it became severe on the 25th. The men were removed into tents for the day, near the parade ground, sleeping in their barracks at night, on the 30th. The disease continuing severe, 97 cases and 72 deaths, on the 8th August, the regiment was marched into a camp on high ground, about four miles off. There was considerable improvement for several days; but the disease again increased, until they changed ground to the opposite side of cantonments, on the 18th August. In doing this they passed through the station, and slept for two nights in their barracks. On the 19th and 20th, there were 17 cases and 14 deaths. The camp then changed ground every third day, and the disease rapidly subsided, and disappeared on the 14th of September. Out of a strength of 1,166, there were 209 cases, and 158 deaths.

The measures of removal, which I had advised and thus carried out, were guided by the principle that cholera is communicable from the sick to the healthy, either directly or

indirectly, by vitiating the locality in which they reside; and the remedy consists in removing the healthy from that locality and isolating the sick. This is effected in the most thorough manner by removal into tents, and afterwards by frequent changes of ground, to short distances, until the disease entirely disappears. Special hospitals for cholera patients are necessary. Removal was thus effected in seven epidemics under my direction with successful results, notwithstanding the season of the year. It was only necessary to avoid marching the men on a rainy day, and guard against direct exposure to the sun in the hot season. The epidemic of 1861 was very general in the North-west Provinces and the Punjaub; and the mortality amongst the European troops extremely high. A special commission was therefore appointed by Government, to investigate and report on the subject, of which Mr. (now Sir John) Strachey was president. They made a minute and searching investigation, and the result was that they approved the measures of removal that had been adopted at Agra; and recommended the issue of regulations enforcing the same in future, on the appearance of similar epidemics amongst the European troops in India. These regulations were much objected to in the years that followed, by several parties; and in 1868 a special commission was appointed at Simla to revise them, of which I was a member.

The medical objections urged before the commission were that removal was needless, as the disease was not contagious; and that it was dangerous, as living in tents during the hot and rainy seasons was likely to induce other diseases, as sun-stroke, fever, and dysentery, which are as fatal as cholera; and to which the men are less liable in barracks. In answer to these objections it was stated that no increase of sickness had hitherto been induced by removal when properly conducted; that, on the contrary, there had, in many cases, been a decided improvement in the health of the men, and their health subsequently had been better than that of the men who had remained in cantonments. Objections were also urged against removal by the military authorities. They did not approve of the responsibility of taking this measure resting with the medical officers; but, as it is a medical question, it was thought that that department was best qualified to form an opinion of its necessity. Other objections to removal are of great practical importance. It gives great and serious trouble to executive officers, who have to make the arrangements. The officers with the regiment suffer much inconvenience and discomfort in

moving from their houses into tents in the hot and rainy seasons. The liability to be recalled from temporary leave is also disagreeable.

These are, doubtless, all real objections; but when the measure in question is for the benefit of the men, they must be set aside. The expense of removal is certainly considerable, but that is more than compensated for by the saving of life among the soldiers.

A return of the instances of removal that had taken place the previous year (1867) shows that out of 198 instances, in 118 there had been no case of cholera after removal, and in only 59 instances had there been any case after the fourth day. The deliberations of this commission were followed by the revised regulations published in General Order of the Governor-General, No. 129, dated August 3rd, 1870, and will be found in the Sixth Annual Report of the Sanitary Commissioner with the Government of India. They differ in no essential point from those previously issued; but are more complete in details, which further experience had proved to be useful. These details comprehend the strictest attention to all sanitary and hygienic rules; enforce the selection of high and well-drained ground for camp to be frequently changed—to be if possible near trees—and if the troops could be taken across a river, the advantage is great.

The injurious effects following deviation from these regulations, indicate the soundness of the principles on which they were founded. The most prominent sources of danger being: first, delay in removal, arising from the non-recognition of the disease; second, deficiency in tents or their transport; third, remaining too long in the same camp; fourth, low damp site for camp, in the rainy season; fifth, exposure to the sun; sixth, too long marches; seventh, occupying ground previously vitiated by the presence of a cholera party; eighth, inattention to the treatment in the preliminary or early stage of the disease. This may be considered a valid excuse when the surgeon does not recognise the disease previous to collapse; but here the defect lies in the surgeon, not in the regulations.*

* In India the disease is present every year, and these regulations are in constant use. Dr. Cuningham, in his last Sanitary Report for 1878, states, page 73: "The medical officers are almost unanimous in the opinion that the evacuation of the affected buildings and movement into camp, when this more decided measure was required, was attended with the best result. In very many instances, no further case occurred. With few exceptions, it is said the change into camp did not induce other diseases; in some cases it is reported, on the contrary, to have had a beneficial effect."

The total saving of life during the sixteen years that removal has been practised has been 18,152, as shown in the Table. The mortality among the European troops in eleven of the chief military stations was 14.90 per 1,000 previous to removal, and 6.69 per 1,000 subsequently. In a financial point of view the lives of European and native soldiers and prisoners are not considered of equal value—the European soldier being estimated at £200, whilst the native prisoner is a negative value; but in a professional point of view all lives are equal.

The diminished mortality is considered by some as the result of sanitary measures, which have been in progress since 1848. The benefit of these is indicated in the Table by a diminution of 16.69 admissions, and 3.80 deaths per 1,000 in the period from 1848 to 1863, as compared with the previous division of time. But the diminution from 1863 to 1878 shows an improvement of 12.75 admissions, and 3.00 deaths per 1,000 on that period. The more perfect sanitary measures doubtless contributed to this; but the mortality in some of the individual stations proves that something more than defective sanitation is required to cause the great mortality from cholera; the virulence of the disease appears unchanged, and it is by escaping from the attack by removal that life is saved. It seems to me strange that regulations of a similar character have not been introduced by any of the Governments of Europe, for the benefit of their armies. The disease is not so prevalent as in India, but there have been severe epidemics since 1831, and their return should be anticipated. If such measures were clearly understood and ably enforced, panic would be averted, which is the ordinary effect of the appearance of an epidemic.

Tables Nos. I, II, III, and IV, show the admissions and deaths from cholera in Bengal, the Upper Provinces, Madras, and Bombay, amongst the European and Native troops, the Native prisoners, and the Civil population. The ratio of deaths varies in each class.

The improvement in all these classes since the introduction of removal, as shown in Table No. V, contrasts favourably with the continued severity of the disease in the civil population, Table No. IV, where removal cannot be carried out as with troops and prisoners.

Other tables bearing on the general question of cholera were also shown, but are omitted here.

TABLE V.

Admissions and Deaths from Cholera among the European and Native Troops and Native Prisoners from 1817 *to* 1878. *From the Records of the Medical Board.*

European Troops—Bengal, Upper Provinces, Madras, and Bombay.

Date.	No. Years.	Strength.	Admissions.	Deaths.	Ratio per 1,000 Admissions.	Ratio per 1,000 Deaths.	Deaths, Admissions.	Saving. Admissions.	Saving. Deaths.
1817 to 1832	16	431,014	16,975	4,130	39.38	9.59	24.33		
1833 to 1847	15	464,939	13,066	5,082	30.23	10.93	38.88		
1848 to 1862	15	783,834	14,199	5,252	18.11	6.46	36.99		
1863 to 1878	16	917,833	4,921	3,177	5.36	3.46	64.56		
Total....	62	2,597,020	49,161	17,638	18.88	6.79	35.88		
1817 to 1862	46	1,679,787	44,240	14,464	26.34	8.67	32.43		
1863 to 1878	16	917,833	4,921	3,177	5.36	3.46	64.56	19,164	4,791

Native Troops—Bengal, Upper Provinces, Madras, and Bombay.

1817 to 1832	16	2,294,709	32,387	9,479	14.11	4.13	29.26		
1833 to 1847	15	2,845,626	25,279	10,089	8.90	3.54	39.91		
1848 to 1862	15	2,639,024	14,039	5,745	5.37	2.11	40.92		
1863 to 1878	16	1,599,512	5,997	3,143	3.75	1.96	52.41		
Total....	62	9,378,771	77,702	28,456	8.28	3.03	36.62		
1817 to 1862	46	7,779,359	71,705	25,313	9.23	3.25	35.30		
1863 to 1878	16	1,599,512	5,997	3,142	3.75	1.96	52.41	8,765	2,127

Native Prisoners—Bengal, Upper Provinces, Madras, and Bombay.

1833 to 1847	15	564,184	21,948	9,288	38.90	16.46	42.32		
1848 to 1862	15	818,770	23,408	10,208	28.71	12.47	43.61		
1863 to 1878	16	1,315,548	16,812	7,356	12.77	5.59	43.76		
Total....	46	2,768,502	62,168	26,852	22.45	9.70	43.69		
1833 to 1862	30	1,382,954	45,356	19,496	33.59	14.10	35.20		
1863 to 1878	16	1,315,548	16,812	7,356	12.77	5.59	43.76	27,389	11,234
						Total		55,318	18,152

Total European and Native Troops and Native Prisoners in India from 1817 *to* 1878.

1817 to 1862	138	10,842,100	161,301	59,273	14.88	5.47	36.76		
1863 to 1878	48	3,832,893	27,730	13,676	7.23	3.57	49.32	28,960	7,283
Total....	186	14,674,993	189,031	72,949	12.88	4.97	38.59		

Deaths and Admissions from Cholera among the European Troops in 11 *of the principal Stations in the Bengal Presidency from* 1817 *to* 1878.

1817 to 1832	16	174,975	6,035	1,909	34.49	10.91	31.46		
1833 to 1847	15	172,158	6,470	2,357	37.58	13.63	36.42		
1848 to 1862	15	280,721	9,519	5,024	33.55	17.90	52.83		
1863 to 1878	16	255,804	2,541	1,705	9.93	6.64	67.10		
Total....	62	883,658	24,565	10,995	27.91	12.44	44.76		
1817 to 1862	46	627,854	22,024	9,290	35.95	14.80	42.18		
1863 to 1878	16	255,804	2,541	1,705	9.93	6.69	67.10	6,425	2,074

ON CERTAIN POINTS CONNECTED WITH THE EPIDEMIC OF PLAGUE IN THE PROVINCE OF ASTRAKHAN, RUSSIA, IN THE WINTER OF 1878-79.

By J. F. PAYNE, M.D., F.R.C.P.,
Late British Medical Commissioner to Investigate the Plague in Russia.

[*Read: December 3rd*, 1880.]

In asking your attention to certain points connected with the late epidemic of plague in the south of Russia, I must beg leave to trouble you first with a short account of the country in which it occurred, since this district is both very imperfectly known, and, at the same time, its physical features are not without importance in relation to the history of the epidemic.

The district in question may be defined, geographically, as including the banks of the Volga for about the last 300 miles of its course. It may be conveniently bounded on the north by the town of Tzaritzin, where the Volga, after approaching within twenty miles of the Don, makes a sharp turn to the east, and somewhat alters its character, forming below this point a larger number of collateral channels on its left bank. A few miles below Tzaritzin, near the German colony of Sarepta, the river leaves the range of hills which, north of this, have accompanied its right bank, and no longer presents in so marked a degree that striking difference between the right and the left bank, which has always been noticed by travellers in the upper part of its course. The physical features of the district generally are uniform. On either side the river is a level, treeless plain, covered in the summer with scattered pasturage, and dotted with a few salt lakes—relics of an evaporated inland sea. The soil is chiefly sand, some places a light clay, seldom suitable for brick-making. It is deficient in lime, and strongly impregnated with salt and nitre. There are scarcely any springs of water, and near the river none at all. The last important land-spring which we passed in going southwards, was at the German village of Sarepta, where the hills are last seen near the river. Even these hills are, at least in this neighbourhood, com-

posed entirely of sand and alluvium, without even a backbone of limestone or rock of any kind. The right bank of the river, though less elevated than in the upper part of the river, is for the most part steep, and reaches at some points a height of more than 100 feet, at others descending to the water level. The left bank is uniformly low. On this side, too, the river breaks up into a number of collateral channels, united by cross passages or canals, which are said to be extremely variable, altering in situations and depth almost every year. The most important of these is the river Achtuba. The district, thus intersected with shallow watercourses, has the reputation of being extremely unhealthy. Some German colonists formerly attempted to settle there, but were decimated by disease, induced by the climate. It owes this character to the annual overflow of the river, which will be described later on. The climate of the district shows great extremes. The winter is severe, though shorter than in northern or middle Russia. The Volga is firmly frozen over for about three months, usually becoming closed in November, and opening again in March. Great variations are, however, observed—the hard frost being frequently interrupted (except in mid-winter) by storms and rain. The winter of 1878-9 was considered exceptionally mild, much rain falling in December; but the early spring was extremely cold, so that the ice of the Volga did not break up till April. Very low temperatures, such as from 30° to minus 19° Fahr., are said to be frequently recorded in ordinary years. In the summer the heat is very great—a temperature of 92° Fahr. in the shade being said to be not uncommon, and very often exceeded. The summer temperature may also be estimated by the fact that the water melon is cultivated, and ripens through the whole of the district. In the spring and summer drought is the chief thing to be feared. During the five weeks which we spent there, not a drop of rain fell, and this appears to be usual at that season. This year (1879), we hear, from later accounts, has been unusually dry, scarcely any rain having fallen during the whole of the summer, so that near Tzaritzin the crops are said to have perished on the ground. The most important physical event of the year is the rising of the Volga. This river, like the Nile, annually overflows its banks, or at least chiefly its left bank, and inundates a considerable tract of country. The annual rise begins at Astrakhan, towards the end of April. We saw the first signs of it in our passage up the river, on our home journey

about this time. The rise continues to the beginning of June, when, after remaining a few days at its height, the river gradually subsides, and regains its old level by the end of June, or the beginning of July. At Tzaritzin, the vertical rise may be from 25 to 30 feet, or even more; but at Astrakhan it is much less, seldom exceeding 6 feet; the difference being attributable to the greater united area of the several branches of the river at the lower point. The breadth of the belt of land, covered by the annual inundations, varies at different points. Near Vetlanka I was told by the mate of the steamer, that the river, in flood, sometimes appears to be twenty miles wide. As these inundations extend for at least 200 miles, and as there is no high-land on the left bank to stand in the way, it is evident that a very large area is thus submerged. The land thus watered is too damp to produce grain, but offers rich pasturage for cattle. We found it, in the very dry weather, before the rise of the river, already green and damp, intersected with swamps and patches of reeds, and still sprinkled with fresh-water shells from the last year's inundation. The above details respecting the physical features of the district may seem tedious. I have, however, mentioned nothing but what has some bearing on the history of the plague.

With respect to the social features of the province, it is to be noted that the Russian inhabitants occupy only a narrow belt of land along both banks of the river. Their villages are dotted at intervals along the post road. But there is a large Tartar population, Kalmucks, on the right bank, Kirghiz on the left, scattered over the steppes, and occupying a few small settlements near the Russian villages. There is a striking absence of scattered houses or farms. From one post station to another, scarcely a human habitation is to be seen. In the villages, consequently, the population is dense. The houses are built of wood, very firmly closed against the cold, and equally, therefore, against any access of fresh air. The windows are double, and rarely, if ever, opened during the winter. The rooms are well heated by stoves, and when the temperature reaches, as it often does, 80 degrees, the closeness of the atmosphere is suffocating. The floors of the cottages are raised on piles two to three feet off the ground, but this advantage is generally almost neutralised by the lower part of the building being surrounded by a sort of embankment of mud, straw, and lime. The water-supply is always obtained from the river, and, in most cases, directly; but

some of the villages, a little removed from the river bank, draw their water from large ponds or reservoirs filled by the annual inundations. There are, generally speaking, no latrines or receptacles for sewage of any kind—all refuse material being simply thrown on the surface of the ground. In their internal arrangements, the houses were far cleaner and neater than we had been led to expect from what we had heard in St. Petersburg. I find that old travellers of the eighteenth century report that the Cossacks were then cleaner in their dress and habitations than the Muscovites or Northern Russians; and as the Russian population of the Lower Volga is entirely Cossack, this remark would appear still to have some weight. The food of the people is chiefly rye bread, vegetables, and fish. Meat is little used. During the long fasts of the Russian Church, even this diet is much restricted. Their drink is tea and coarse spirit, made from corn. Excessive indulgence in the latter is certainly, so far as our observation went, rare; and if this district were a fair example, I should be disposed to think that the reports of the intemperate habits of the Russian peasantry were somewhat exaggerated. But in this point, too, there is probably a difference between the North and the South.

The only other important fact bearing on the sanitary condition of the villages is the process for preparing and curing the fresh-water fish, which are caught in enormous numbers, and which constitute the chief source of wealth to the population. These fish are cured in tanks filled with brine, which are collected in large establishments called Watagas. It is said, that in consequence of the high price of salt, the brine is used over and over again, till it becomes loaded with organic matter, and is in summer putrid and highly offensive. This statement we were unable to verify, since the season of fish-curing had not commenced; and at the time of our visit, such Watagas as we visited had nothing offensive. It is impossible, however, to discredit the precise statements, both of Russian and foreign travellers, that in summer a Wataga, in full activity, poisons the air for hundreds of yards around, so that in descending the river, the existence of one of these factories is often recognised by the nose long before it is in sight. It also appears that no place is reserved for disposing of the offal of the fish, which is simply turned out with other refuse on the neighbouring land. The work of the Watagas is done by men and women. The latter are hired in gangs by contractors, and it is said that the same moral evils result, as have been

observed in other places where women's work is done in mixed company, and often far from their homes.

Vetlanka, the village which bore the chief stress of the epidemic, was the only one we saw where a Wataga was situated in the middle of the village. This establishment was destroyed after the epidemic by the Russian authorities. It should also be stated that the houses are closely packed, showing in Vetlanka itself an average of nearly six persons to each house.

The history of these villages is not without importance. At the end of the last century the district was almost uninhabited. Tzaritzin was originally only a fortress, and two other minor fortresses, Tcherny-jar and Enotajewsk, were first built. Afterwards, in place of garrisons, military settlements called Stanitzas were founded, and of these Vetlanka is one. The whole of the male population of a Stanitza is liable to serve in the Cossack irregular forces, and not in the regular army. They are not governed by a village council like other Russian villages, but are under the control of a General who is stationed at Astrakhan. There is also a special medical service belonging to the Cossack organisation of the Stanitzas. The other villages are governed and hold their land under the ordinary Russian communal system. The German Moravian settlement (Sarepta) belongs to neither of these classes, but has an independent administration, subject only to a government department in St. Petersburg. The last epidemic of plague known in the district was in the year 1807, when it prevailed in Astrakhan, being thought to have come from the Caucasus, and penetrated to a village forty-seven miles from Sarepta, but no further. The name is not given in the account from which I derive this fact, but I believe it to have been Tsaref. The population at that time must have been very much thinner than it is now, and many of the existing villages were not built. A quarantine station was at that time set up in Sarepta, and was not finally removed until 1823.

I must now speak of the history of the last epidemic, but must say beforehand that this is even now imperfectly known, as is, indeed, confessed even by the Russian medical authorities. Dr. Reutinger who was placed in medical charge of Vetlanka after the epidemic, writes as lately as May 31st, 1879, in the St. Petersburg *Medicinische Wochenschrift*, "Unfortunately we possess no exact reports on the epidemic of plague in Vetlanka. The short memoirs of Drs. Doppner and Rutkowsky appear to have been written more under the influence of other classical descriptions of

the plague. Of the three doctors who died only one, Dr. Morosow, left one history of a case with the day of the commencement of the disease and of death, and gave the diagnosis—pneumonia crouposa. And the statements of Dr. Krassowsky, which I read in Vetlanka, are also superficial, and point only to inflammation of the lungs as a prominent symptom." It may also be remarked that Morosow arrived in Vetlanka when the epidemic was at its height, and Dr. Krassowsky at the end of it.

Dr. Reutlinger wrote thus after spending some months at Vetlanka, so that if we have not now got complete information on the subject, it is to be feared we never shall. To make it clear, I should say that the three doctors mentioned, who died of the plague, were Cossack medical officers sent by the General of Cossacks at Astrakhan. Dr. Döppner, whose report is, I think, too slightly spoken of by Dr. Reutlinger, was the superior medical officer of Cossacks. He visited Vetlanka at the beginning of the epidemic, and again at its end; as his report has been published in several journals, both on the Continent and here, I need not specially refer to it. It seems, however, that the epidemic in the village of Vetlanka began in Oct. 1878 (Döppner says November), and was at first comparatively mild—the disease consisting of fever accompanied by buboes, and not causing any great mortality. It was only about the beginning of December, and, it is said, suddenly, about the 8th of that month, that it assumed the more formidable character of a plague epidemic. The mortality became much greater, thirty or forty persons dying daily, and the course of the disease was more rapid, carrying off its victims after one, two, or three days' illness. At this time congestion of the lungs and spitting of blood became prominent symptoms, and buboes either did not occur or were not observed. This extreme severity of the epidemic lasted till about the 1st of January 1879, when it began to decline. This remarkable implication of the lungs has not lately been observed in epidemic plague, but recalls the descriptions given of the Black Death of the fourteenth century. It was, perhaps, for this reason that the doctors, who first observed the disease in Vetlanka, hesitated to call it plague, but described it under the name of epidemic pneumonia, or galloping typhus. Even those who were most sceptical at first, agreed in the end that it must have been Oriental plague (Döppner called it "Indian plague"). After the decline of the epidemic in Vetlanka only one or two sporadic cases were observed there, the last of which we saw

when already convalescent. The disease appeared, however, in several other villages.

In these places it showed the character of the later, not of the earlier form of the Vetlanka epidemic, and as buboes were in some places never observed, some doubt was felt as to whether the disease here was truly plague. Symptoms alone scarcely distinguished these cases from very severe typhus with inflammation of the lungs. But looking at the whole history, it seems impossible not to regard it as the history of a single disease.

Time will not permit me to enter on several interesting questions connected with the spread of the disease in the district. I shall, on the present occasion, confine myself to discussing, what is perhaps the most important point as bearing on the conveyance of disease from one country to another, viz., the question: *How did the plague arise on the banks of the Volga?*

Theories are not wanting, but two at least seem to deserve special notice. According to the view at first favoured, the disease was introduced from Resht in Persia, at the southern side of the Caspian Sea, where plague is known to have prevailed in 1877, and at the beginning of 1878. There is no doubt a considerable trade existing between Resht and Astrakhan, and on a small map it looks very possible that the disease should in this way have been brought to the banks of the Volga. It can hardly, however, be supposed that the infection was carried direct from Resht to Vetlanka. When we consider that this village is 130 miles from Astrakhan, which is further separated from the Caspian itself by the delta of the Volga, making perhaps another 70 miles, the theory of direct importation to Vetlanka, a distance altogether of 800 miles, becomes extremely improbable. It is as if a disease, supposed to be introduced into the Port of London, should first be heard of at Oxford or Bath. Moreover, there is no reason to believe that plague existed at Resht in the autumn of 1878. Those, indeed, who believe the Vetlanka disease to have come from Resht, now base their opinion upon the alleged occurrence of the plague a year before at the intermediate point of Astrakhan. It is, then, supposed that the plague crossed the Caspian in 1877, and in the next year made its way further up the river.

Another theory, favoured by the head of the Russian Commission, Professor Eichwald, has also received the adhesion of Professor Hirsch of Berlin. It is that the plague was introduced by Cossacks who had served in the Russian Army of Asia, when returning to their homes on the Volga. It is

supposed by some that this disease prevailed (though not recognised) in the Russian Army itself; by others that it existed among the Turks, though without infecting their Russian conquerors; and, again, a third supposition is that some of the infected articles coming from Bagdad, or some part of Turkish Arabia, passed through the hands of Turkish and Russian soldiers without communicating the disease, and gave rise to it only when brought to the homes of the latter in Vetlanka.

Beside these two views, it is also right to consider the possibility of a spontaneous origin of plague in the district itself—a possibility which the physical features of the district strongly impressed upon my colleague — Surgeon-Major Colvill. It may also be thought that the condition of the country and its inhabitants, if they do not account for the origin of the plague, at least show that, however introduced, it found here a congenial soil.

Before proceeding further, I will thus sum up briefly the circumstances which appear to make the banks of the Volga a suitable home for plague.

The banks of a great river liable to inundations have often, as we see in Egypt and in Mesopotamia, been the seat of epidemics of this disease. The general character of the marsh land on the left bank of the Volga (which I may remark in passing was not visited by Professor Hirsch or the other German Commissioners), reminded Mr. Colvill of similar country in Mesopotamia. The entire want of any sewage arrangements and the customs of the fisheries cause a large amount of decomposing organic matter to be constantly poisoning the air in the neighbourhood of the dwellings; and the habits of the people, though more cleanly than has been represented, assist in maintaining a putrid atmosphere indoors. The range of temperature through the year is just what is found to be most favourable to plague, a disease which requires summer heat for its development, but ceases when this heat becomes extreme. Again, although the villages on the right bank are mainly situated on high ground, there are scattered dwellings and villages in the marsh-land on the left and in the bed which must be constantly damp. Here we saw underground dwellings; which must be, in such a soil, among the most unhealthy of human habitations. Such an one we saw at Kapitanski, and others are described as being the abode of Kalmucks on the islands of the Volga. The objection has been made to drawing any deduction from the insanitary condition of the Russian villages, that the Kalmucks live in a far

worse physical condition but are not known to have suffered very greatly from plague. Five Kalmucks, however, died on an island of the Volga, a part of the marsh-land such as we have described, and others were found to have suspicious scars of buboes. This led to an examination of the Kalmuck priests, some of whom were also said to have scars of old (perhaps some years old) buboes. An examination of the Kalmuck population was suggested but not carried out, but I learnt from some who had gone carefully into the question, that so little is known of the intimate habits of the Kalmucks that it is impossible to say whether the disease did or did not prevail among them. Moreover, in the three places which suffered most from the plague, there was in each some sanitary defect: in Vetlanka there was a Wataga in the middle of the village, from which the women would go home their garments soaked with putrid brine; Prishib is very near Vetlanka. In Selitrennoi we traced a close connection with a farm district on the marsh-land, and in Staritzkoi we observed that part of the village was really in the marsh and that the water-supply was from stagnant pools. And in all cases there must have been more or less communication with the belt of unhealthy country along the Volga bed.

To go back to the theory of importation of plague by the Cossacks we see here that the special incidence of the disease on Vetlanka might be to a slight extent explained by the fact that Vetlanka is one of the Cossack Stanitzas. On the other hand there are serious objections to this view, which were shortly pointed out by Surgeon-Major Colvill and myself in our report on the plague in Russia. To begin with, it is an obvious difficulty that the soldiers of the army of the Caucasus, with their spoils, were distributed to many parts of Southern Russia, and not to this district only, though it is only here that plague is said to have occurred. This objection was, indeed, put forward by Professor Hirsch himself, in a lecture delivered before his departure for Russia, though after inspection of the country he seems to have found stronger reasons which outweighed this. Since our report was published I have seen a paper by Dr. Reutlinger, formerly of Vetlanka, in which he states this objection very strongly, and with further details, pointing out that the regiment called the Moscow Grenadiers, which suffered severely from fever at Kars, returned to Russia in April 1878, without bringing the plague with them; the fortieth division of infantry, which was concerned in the siege of Erzeroum, came back in September of the same year to Charkow; the reserve division still later; and that

even the Astrakhan Cossacks were distributed through all the stanitzas of the Governments of Astrakhan and Saratov; but that nowhere, except in Vetlanka, did plague break out. It is not absolutely impossible, that out of a thousand seeds only one should take root; but we should require very clear evidence of the actual existence of such a seed, and proof of its alleged origin.

Again, other questions arise. (1.) Is there really any ground for believing that anything like plague existed in the Russian army of Asia? (2.) Did it exist in the Turkish army, or among Turkish prisoners, or in the population of the Caucasus? Can any articles whatever, found at Vetlanka, be proved or even strongly surmised, to have come from any place where plague existed? (1.) The only plausible reason for suggesting that the Russian soldiers suffered from plague, is the statement that a disease called bubonic typhus prevailed among them, and might have been plague under another name. We enquired of several medical officers of great experience, who served in the Asiatic campaign, and they unanimously asserted that they never heard of such a disease as bubonic typhus in the army. Typhus, typhoid, and relapsing fever were prevalent, and might have been occasionally accompanied by swellings of the parotid glands, but not of the lymphatic. Dr. Reutlinger, who visited, as Government Inspector, many of the army hospitals, made special enquiries in consequence of the fear which prevailed at Tiflis after the campaign, lest the plague, which was known to exist in Persia, might find its way there; but he could not find a single case of enlargement of the lymphatic glands. Further, we must remember that the Russian doctors serving in the Asiatic campaign, were well acquainted, at least by report, with all the symptoms of plague, and constantly on the watch against the possible introduction from Persia or Turkish Arabia. The very notion of bubonic typhus, or pseudo-plague, having existed in the army, did not arise till long afterwards. It is the less necessary to say more about this, because Professor Hirsch himself rejects the theory of plague having actually existed among the Russian troops. (2.) There is no better reason for thinking that the Turks, when in the field or prisoners, suffered from plague. Dr. Reutlinger and others visited the Turkish hospitals, both at Kars and Erzeroum, and found no evidence of plague there. None of the Turkish army doctors have, so far as I know, ever admitted that they saw a case of plague in the campaign, although the disease is well known in Turkish Armenia and Arabia,

both to the doctors and to the non-medical public. We have, again, very precise statements regarding the Turkish prisoners in Russia. In the winter of 1877-8, the Russian Government appointed a Special Commission to enquire into the alleged occurrence of fever among the Turkish prisoners. The Commission visited almost every place where prisoners of war were living—both those who were taken in the European and in the Asiatic campaigns. Dr. Metzler, one of its members, states that he found the three forms of typhus (as they are called by the Germans and Russians), dysentery, and other diseases, very prevalent, but was never able to detect the least sign of plague, nor had any of the doctors in charge of the prisoners seen a case which even gave rise to suspicion. He also points out that most of the prisoners still wore their own, often very ragged, uniforms; and that so long as these could still be used, they continued to wear them. During the year which the prisoners spent in Russia, no case of plague occurred among them, or among any persons with whom they were in contact, and even the fevers from which they actually suffered did not spread to the population. Finally, did any plague or plague-like disease prevail among the population of the Cascasus or Armenia, while the Russian and Turkish armies were there? Of this also there is no evidence. (3.) It seems, then, clear, that if the plague came out of the Turkish army at all, it must have been through some object which did not communicate the disease in transit. Now, it must be remarked that the Cossacks of Astrakhan took no active part in the campaign till after the capture of Kars, in November 1877. After that event, they escorted Turkish prisoners to Alexandropol. It was from these prisoners that they must have obtained, if they obtained at all, the infectious objects. It is stated by Professor Hirsch that the Cossacks sent part of their spoils home to their families before they themselves returned. Let it be so; but these articles, if they came from Bagdad, or any part of Turkish Arabia, must already have been some months on the journey, and before they arrived at Vetlanka, if they contained any infective matter, it must have been nearly or quite one year old. The epidemic supposed to have been caused by them did not begin till nearly a year after they had been in possession of the Cossacks.

Let us now consider what these objects were. The greater part had been burned before we arrived at Vetlanka, but from the account given it is clear that they chiefly consisted of the clothing of the Turkish soldiers. It can hardly

be imagined that these garments could have conveyed to a distance the disease which they had not given to those who originally wore them; but it is said there were also some articles of women's apparel—shawls and silk dresses. Dr. Reutlinger declares that they were much more like the cast-off clothes of Russian ladies, and some were clearly of Caucasian manufacture. We heard in Vetlanka a very precise description of a certain silk dress trimmed with velvet, which, from the account given, was clearly, in Mr. Colvill's judgment, such a thing as is never seen in Bagdad. Whether there was more than one such garment I cannot tell, for we know how in such cases the singular very easily slips into the plural. Professor Hirsch supposes that some packed goods which had been brought from Bagdad to Armenia without being opened, were sent from Armenia to Astrakhan still unopened, and when unpacked in Vetlanka, produced the disease. I cannot find, however, that any such unopened packages can be proved to have existed, and it would seem strange that the Cossacks should send or bring presents to their wives without taking the trouble to open the packages to see what was inside. Again, we should not attach much weight to the belief of the people themselves or of a priest on this subject, were it not that this testimony has been quoted by Professor Hirsch. It appears to me, with all respect to the distinguished authority who has favoured this theory, that it is only right to subject it to very careful scrutiny, and that little short of positive proof should be admitted for a supposition at first sight so improbable. In the absence of such proof it would be better even to leave the question quite undetermined. There is a great danger that a mere hypothesis once adopted by an authority of weight, and accepted as the official statement, may find its place in the history of epidemics as an acknowledged fact.

I have now to consider the connection, if any, of the Volga epidemic with that of Resht in 1877. The apparent intermediate link is to be found in a peculiar disease which prevailed in the neighbourhood of Astrakhan in (June), July and August, 1877. The accounts of this disease are conflicting, and at a distance of two years we can hardly expect to get at a perfectly accurate knowledge of it. Official reports on the subject are, I believe, in existence, but though they have been applied for, no copies have yet been received in this country. It seems, however, that at several villages near Astrakhan, viz., a little place called Vorpost, on the opposite side of the river, a village or district called Kasatchi-Bugor (which I gather to be a general name for

low islands in the delta of the Volga), about 120 persons are believed to have suffered from some febrile affection, accompanied by buboes. Other cases occurred in hospitals of Astrakhan. There was, however, no case of death, or only one doubtful one. This type of disease is known to have been prevalent at Resht in the same year, and has also been reported as occurring in Mesopotamia and Persia, either before or after epidemics of true plague, by Tholozan, Cabiadis, and others. Surgeon-Major Colvill has some materials, collected in Bagdad, bearing on the same subject, but as they are not yet published, I will not further refer to them. A Russian physician, Dr. Janizky, has given this disease the name of *pestis nostras*, and in Mesopotamia it has even received a vernacular name—*chiarek*. Dr. Kastorsky, who investigated the Astrakhan disease for the Russian Government, recognised its identity with the epidemic of Resht. This disease, whatever it was, slumbered for more than a year, but I can hardly myself doubt that it was the same which re-appeared in the earlier and milder stages of the epidemic at Vetlanka. It is true that the connection, though very close, is not quite direct, either in time or place; but if the Astrakhan disease was in any sense plague, it is only in accordance with the history of this malady, that it should lie dormant from one season to another, and when it re-appeared, should be found at a spot somewhat removed from its former habitat.

This appears to me to be the most important, and, indeed, the crucial question which has been raised by the history of the last epidemic. The conclusion to which it appears to point, though by no means to prove, is as follows:—the disease called "plague" would seem to be capable of existing in two forms—the one a mild, non-fatal form, and not easily transmitted from one person to another, in which the lymphatic system is chiefly affected, and the fever holds a subordinate position: the other, extremely malignant, very readily transmitted (whether by contagion or infection may be regarded as uncertain), and in which the fever is of such intensity, that death often occurs with few or no organic lesions. The milder form would be so easily overlooked in countries badly provided with doctors, that it is easy to suppose it to be really endemic, or else continued from one year to another by epidemics so light as not to excite attention. But if we suppose that such a disease, being introduced into thickly populated places, its malignity being heightened, as is the case, for instance, with typhus, by gathering together large

numbers of sick, we should then have an epidemic such as appears to correspond to the general character of plague, as seen in former times in this and other countries. It is easy to understand how this disease should have been overlooked in the best known and most carefully observed epidemics of plague. These have always been such as have occurred in great cities, where the disease, in a highly communicable form, has been introduced, and found a fitting soil. But, to understand it rightly, further and continuous observations are needed in those countries, where it occurs more frequently, and where every degree of transition is found between the mild endemic and the malignant epidemic.

THE REPORT OF THE IMPERIAL GERMAN MEDICAL COMMISSION ON THE PLAGUE WHICH PREVAILED IN THE PROVINCE OF ASTRAKHAN DURING THE WINTER OF 1878 AND 1879.

By J. LAWRENCE-HAMILTON, L.R.C.P.Ed., etc.

[*Read: May 5th,* 1880.]

In the following paper I have endeavoured to give a brief, but accurate, summary of the official report of the Imperial German Medical Commission on the outbreak of plague in the province of Astrakhan during the winter of 1878-9.

In January 1879 news reached Berlin of the appearance of plague in Russian Astrakhan. As both the official reports and information derived from the newspapers on the subject were vague and unsatisfactory, the German Government, acting in harmony with the Governments of Austro-Hungary and Roumania, and with the official sanction of Russia, sent out a medical commission of inquiry to Astrakhan. Germany was represented by Dr. August Hirsch, Dr. Küssner, and Dr. Sommerbrodt; and these gentlemen, accompanied by a professed linguist, Mr. Rössler, arrived in Astrakhan towards the close of February 1879. The Commission write in high praise of the courteous and attentive assistance rendered to them by Count Loris Melikof, the then Russian Governor-General, specially appointed, with extraordinary powers, in view of the plague in the province of Astrakhan. The report, written by Dr. August Hirsch and Dr. Sommerbrodt, recognises the assistance derived in the inquiry, from the Austro-Hungarian Commissioners, Professor Biesiadetzky and Dr. Kiemann; the Russian Commissioner, Dr. Eichwald; and the French Commissioner, Professor Zuber.*

* It may be mentioned that the English Medical Commission did not get to Astrakhan till some three weeks after the German. The British Government selected Surgeon-Major Colvill of the British Residency, Bagdad, and Dr. J. F. Payne, of St. Thomas's Hospital, as commissioners. The Swedish Commissioners were Drs. Duner and Malthe, whilst Den-

Astrakhan is a province of Russia, bordering the Caspian Sea to the north-west, and traversed by the rivers Volga and Ural in the latter part of their course. The right bank of the Volga is some thirty metres or more above the level of the sea, whereas the left side is but little above it, and is liable to frequent overflows from the river during floods. The rich salt marshes of Astrakhan are well known. The province is chiefly a vast steppe or huge extended plain, and almost destitute of trees. The summer dryness is intense, as here heavy rainfalls are most rare and exceptional. With the closing autumn, and with the commencing spring, frightful snowstorms rage, when, owing to the dryness of the soil, such immense masses of dust are apt to be whirled or raised up, as even occasionally to produce a change in the physical appearance of the surface, and indeed quite to choke up with sand an entire neighbourhood. The climate is very hot, often tropical in summer, whilst the cold in winter is very severe. Towards the latter half of November real winter sets in, the rivers become frozen over, whilst the temperature is often some ten degrees Reaumur below the freezing point of water.

The domiciled population of Astrakhan is given at 500,000, whilst 150,000 of the tribes are still nomadic in their habits.

The accumulated observations of twelve years record one birth for every 36·5 persons, whilst one death takes place for every twenty-one of the inhabitants of Astrakhan; or in other words, this population would gradually become extinct were it not being continually recruited from external sources. Grain is only capable of cultivation in very circumscribed localities. Mustard is abundant and remunerative. The Tartar tribes give attention to gardening. The number of oxen, horses, and camels in Astrakhan is estimated from two to two and one-third millions, of which upwards of 40,000 are employed as transport animals within the province. The principal occupation of Astrakhan province is fishing, which alone demands the services of some 50,000 boatmen. Caviar, or the prepared roes of certain large fish salted, and the most celebrated in the world, is largely exported; also fish, isinglass, train-oil and blubber.

mark was represented by Dr. Feilberg. M. Lallemand was a further Commissioner from France, whilst Dr. Jacoby was the second Commissioner in rank selected by the Russian Government.

There was no Joint or International Commission, as the Russian and Foreign Commissioners decided to act independently, though some of them travelled together in company.—J. L. H.

The transport traffic from and to other provinces of Russia, for and from the Caspian Sea, Caucasia, and Asia, and along the River Volga, is extensive. In Astrakhan, for the carrying trade by shipping on the Volga alone, some 6,000 hands are employed in connection with about 70 steamers and 800 sailing vessels.

There is a railway completed between Tzarizin and Griasi. The German Commission dwell deprecatingly on the absence of European consuls at Tzarizin, Persia alone possessing an official representative there.

In Astrakhan province most of the people seem well to do, but a fearful want of general cleanliness is here as strikingly characteristic as in other parts of Russia. In the city of Astrakhan most of the streets are devoid of pavement. There are no fresh-water springs, and the water-supply is drawn from the fouled branches of the Volga.

The labourers employed in fish salting exist under miserable conditions. In many places they dwell in cavities hollowed in the earth, or in caverns. The price of bread being beyond their means, they subsist chiefly on the leavings of the inferior parts of the prepared fish. Formerly government rules enforced that the unused remains of the prepared fish should be thrown directly into the water, but now these, collected and accumulated in masses, are left to rot in and about the banks of the rivers under the heat of sometimes an almost tropical sun. Further, the vats used for salting the fish are never properly or systematically cleaned. It is the custom merely to add more salt from time to time. The local atmosphere is further vitiated by many fat-boiling works.

The German Commission does not attribute the outbreak of the plague directly to these local conditions, as for many years this disease has not visited these localities. Owing to the general low-lying ground immediately bordering the Volga, malaria, rheumatic fever, inflammatory diseases, chiefly of the respiratory system, are prevalent, though it has been specially noticed that phthisis seems almost unknown in the province. Scrofula and syphilis are reported as being almost universally present.

During the five years preceding the outbreak of plague, enteric fever, measles, and small-pox had been epidemic, whilst scarlet fever raged in 1876 and 1877. During the previous four years the Commission heard only of the occurrence of one case of maculated typhus, but typhoid fever had been comparatively frequent.

The town of Astrakhan during the last twenty-two years

has suffered nine epidemic attacks of cholera, and three of enteric fever. Previous to the last outbreak of plague, the epidemic had not been present in Astrakhan since the beginning of the century. An outbreak of the disease occurred, commencing in August 1807, and continuing to February 1808. Comparatively few persons were then attacked, and the mortality was but small.

In 1878 and 1879 the paucity of doctors, army-surgeons, and medical assistants was lamentable, especially outside the city of Astrakhan. The recent epidemic showed them to be deficient in the art of diagnosis, as they long reported the plague as being "malignant typhus", "pneumonic croup", and so forth.

In Astrakhan province, plague made its first appearance probably during the first half of October 1878; the earlier cases being mild in character and not ending fatally. The official Russian reports couple these first cases with malarial fever, whilst other reports state that plague was superadded to the already prevalent "bubonic malarial fever". We are not able to estimate the truth of either of these statements. It is, however, certain that plague arrived in Vetlanka when the local and native physicians had already spoken of epidemic, malignant, malarial fever, and of enteric fever.

In Vetlanka the plague began either towards the close of September, or else early in October 1878, whilst the epidemic spread generally about the end of November 1878.

During the three terminal weeks of November 1878,* the number of deaths from the plague in this district were 7, 8, and 7 respectively. Whilst in the first week of December there were counted 56 victims, in the second week the maximum number of 169 was reached, and in the third week the number fell to fifty-four. Hence the decline was rapid, and the very last death occurred on the 12th (O.S.) of the following January (1879). In Vetlanka 20 per cent. of the entire population was carried off, or 25.3 per cent. were attacked, with a mortality of 82 in the hundred seized.

The course of the epidemic was highly erratic, now here, now there. It seemed to be influenced and determined far more by personal communication among the families than by geographical or topical considerations.

The public in Astrakhan soon became alive to the presence in its very midst of the plague. Then all at once the community was seized with a panic. Family ties were

* The dates throughout are given in Old Style.

broken asunder, and the natural loyalty of near blood and of old friendships severed. Upon the faintest suspicion of being stricken with the plague, the individual was forthwith thrust out of the community by his nearest relative, into the common pest-house. Beyond doubt, many a one afflicted with other and trivial diseases, by being flung into the pest-house, caught the plague. All shunned the house where the plague had entered, and hence several little helpless children were left within to die of starvation.

Among other instances may be noticed that of a brave local priest who conscientiously nursed and tended the plague-stricken of his flock. He himself soon succumbed to the plague. No one could be induced to bury him. So his wife, though far advanced in pregnancy, aided by her sister, dug out his grave in the hard frozen ground. A few days afterwards both these women were dead from the plague.

A young woman, eighteen years of age, having complained of pains in the head, was thereupon immediately incarcerated in the pest-house, where drunken nurses could only be obtained. (Indeed the people believed that it was only safe to attend upon the sick, when the nurses were under the influence of brandy.) She soon fell ill with the prevailing epidemic. Upon recovering consciousness she discovered herself in a room with seven corpses, where the windows of the apartments were broken. It was now the depth of winter. To keep out the cold she was compelled to seize upon the clothes and bedding of the dead around her, yet both her feet suffered from frost-bite. Becoming somewhat stronger, her continued shrieks brought her relief from her captivity through a man, a stranger, chancing to pass by.

The doctors and nurses becoming so very rapidly fatally ill, the extent of the panic increased.

Continuing along the course of the Volga, Prischib was next visited by the plague. On November the 30th (O.S.) 1878, from Prischib, one Irina Jeffframowa, drove over to see her relations in Vetlanka, among whom was her soldier-brother, who had returned from the wars, and in whose house she resided. On December 3rd she went back to her home in Prischib, where she was taken ill on the 5th, and died on the 11th of the month. Her household chanced to consist of nine persons, all of whom, save her mother, were dead by the 20th.

From Prischib, three sisters of mercy stayed in Vetlanka *for only twenty*-four hours, and were again home by the

8th of December. On the 12th they were attacked by the plague, which carried them off by the 16th. Six days later a female servant in attendance upon them next succumbed. These sisters of mercy had been attended to and buried by four nurses and carriers of the dead, who lived next door, and who also were taken ill, and all were dead by the 24th of December (1878).

In five houses in Prischib, between the 5th and the 24th of December, there occurred sixteen deaths, including two children.

Through personal communication Vetlanka was evidently the infecting focus to several adjoining districts.

The Vetlanka outbreak commenced in the first half of October 1878, and closed with the end of January 1879, after having sacrificed 420 victims. Nine of the civil and military doctors stationed in and about Vetlanka died of the plague.

Dr. Rudkowski, who saw the last four cases which occurred in Staritzkoie, gives the following account of the case of a strong-built girl of nine years of age.

On December 29th the child was suddenly taken ill at three in the morning with cold shivers, headache, thirst—persistent even after taking cooling drinks—and constipation. At nine on that morning Dr. Rudkowski saw the child. She was still able to sit on a bench. Her face was pallid and puffy; the temperature above the normal; the respiration free; no rash present. She complained of pain in the abdominal region and of severe headache, was very thirsty, and had no appetite.

2 p.m.—Dr. Rudkowski observed that her tongue was rather dry and coated with white fur. The pulse numbered 100, but was small. The constipation continuing, castor oil was administered.

7 p.m.—The girl's bowels had been opened. She now vomited after each attempt at drinking, and refused to take any further medicine. She complained of headache, and of oppression about the pit of the stomach. The tongue was white, the respiration still free, but the temperature increased.

December 30th, 9 a.m.—The patient had passed a bad night, and was suffering from thirst and a feeling of general anxious oppression. She vomited after taking physic. Her temperature was 37° Centigrade. No urine was saved. No further motions had been passed.

2 p.m.—The girl was throwing herself about violently, as she lay on the ground, chiefly on her belly, with her feet

outstretched. She complained of violent pain and great oppression. The pulse was accelerated. No urine passed. Spasms had been observed in the hands and feet.

7 p.m.—Dr. Rudkowski found his little patient wandering and shrieking, whilst the nurse reported tetanic-like spasm "with arching of the spine and extremities". Later in the night these attacks are stated to have occurred. On the 31st of December death took place, after an illness of forty-eight hours' duration. Dr. Rudkowski found the girl dead, lying on her abdomen with outstretched legs. No spots or swellings were observed after death.

In other fatal cases Dr. Rudkowski observed that the jaws were firmly set or closed, and the elbows bent. It is noted that his observations were neither taken down at the time nor at the bed-side, but given later from memory. Dr. Rudkowski thus describes the broad course and symptoms of the plague in the cases he saw:—It began with a sense of cold, headache, thirst, and in a few hours later with a dreadful feeling of peculiarly grave oppression, which Dr. Rudkowski considered as pathognomonic of plague. Consciousness remained save during the attacks of tetanic cramps. The patients were themselves fully aware of approaching death. Dr. Rudkowski did not observe in any of his patients the presence of glandular swellings, nor any rash on their skin. Constipation was general. The urine was at first scanty and then suppressed. Dr. Rudkowski took the temperature thrice daily. It generally varied between 37 deg. and 39 deg. Centigrade; in one case only did it amount to 40.5 deg. Centigrade.

In a few cases anæsthesia was observed, pricking by pins not being felt.

The condition of the spleen and liver was not examined.

In every instance death took place within forty-eight hours after the manifestation of acute symptoms.

Dr. Rudkowski examined his patients with his hands previously anointed with carbolised oil. He disinfected his hair and beard with the carbolic acid spray. *Post mortem* examinations were not made.

In Vetlanka several cases were reported in which buboes were found in the armpits, groins, and one instance in the right side of the neck. Where recovery took place, suppuration of the buboes occurred occasionally. The German Commission fully believe that the disease was the bubonic plague.

In Vetlanka, indeed, the epidemic was distinctly bubonic, with the comparatively small fatality of 50 per cent.,

whilst in Prischib and Starizkoie the disease was unattended with buboes. Possibly, at the commencement of the epidemic, in the intense fright and panic, the existence of buboes might have been overlooked. The German report lays stress upon the unity of the two forms of plague—that with buboes, and that without buboes. For example:—

The family of Charitonow comprised three living generations, numbering thirty-two individuals, of whom twenty-five being seized by the epidemic, thirteen died, nearly all of whom had buboes.

Ossip Bielow's household counted seventeen souls, all of whom succumbed to the plague. It is reported that not one of these had any buboes.

The Turkish Medical Commissioner, Dr. Cabiadis, who had observed the last plague epidemic in Mesopotamia, remarks that, in the agony of the plague, though small buboes may be now and then overlooked, in the most severe and rapid cases of plague there is no time for the development of the buboes.

The official lists of the sick and dead, as reported by the Astrakhan doctors, were kept in such a slovenly, irregular way as to become practically worthless. Thus, from a heap of errors, in a report signed by two of the local doctors, among other blunders, three people were returned as having died of "pneumonic croup", who were afterwards seen to be alive and well by the German Commissioners. The European experts found, among eighty-one convalescents from the recent plague, fifty-one persons quite free from scars of buboes or glandular swellings, eleven scarred with healed buboes, in eighteen the remains of glandular swellings not yet dispersed, and in one case an ulcerated bubo.

Of the eleven presenting scars of buboes, five of the scars were situated among the crural glands, three among the inguinal glands, two were situated under the jaw, and one was located in the armpit—an order of occurrence, by the way, which agrees with that given by Griesinger.

The buboes are reported often to have attained to the size of a walnut, and to have been generally solitary. It was most exceptional to find both sides of the body thus affected; it was still rarer to see two buboes in different regions of the same patient. Buboes, when opened, either by art or by nature, were found to afford great relief to the patient. From the third to the sixth day or later was the usual time for the bubo to mature. Convalescence was generally ushered in and indicated by profuse sweating.

There appears to have been a bare possibility that the plague may have been preceded by lung complications.

The Vetlanka epidemic began with the severest form, terminating generally in death within forty-eight hours. The minimum period of incubation would seem to have been two days and a-half, and the maximum eight days.* The average period of latency of the disease was 5.2 days, though in acute cases a shorter interval sufficed.

According to the views of some, plague in Astrakhan was derived from the Persian town or neighbourhood of Resht, which is but a few miles from the southern border of the Caspian Sea. In this city it will be remembered that plague raged virulently from November 1876 till October 1877. Now it happened that in the summer of 1877, the town of Astrakhan and its vicinity were afflicted with a malady, which may possibly have been a mild form of the disease.

The malady in question is described as being complicated with various glandular swellings in the axillary, submaxillary, inguinal, and crural regions. Long protracted fever was present. The swellings seldom attained to a large size, and terminated as a rule in resolution. Only one case proved fatal, pyæmia being stated as the cause of death. In the town of Astrakhan probably some 100 to 150 persons were attacked by this disease. The garrison of the city was not attacked by it, and only one instance of contagion occurred among the nurses and attendants upon these sick persons in the summer epidemic of 1877.

* The following table is given relating to the period of incubation :—

	Incubation Period in Days. Minimum.	Maximum.
From Staritzkoie.		
In 1st case	2	3
„ 2nd „	1	3
„ 3rd „	4	6
„ 4th „	5	7
From Prischib.		
In 5th case	2	?
„ 6th „	3	9
„ 7th „	3	9
„ 8th „	3	9
„ 9th „	3	9
„ 10th „	2	14
„ 11th „	1	15
„ 12th „	1	15
„ 13th „	4	?
„ 14th „	4	?
„ 15th „	?	4
„ 16th „	4	?
„ 17th „	1	8
„ 18th „	1	5
„ 19th „	2	7
„ 20th „	2	7

The clothes of all who fell sick in 1877 were disinfected by the authorities in the hospitals of Astrakhan.

As has been previously observed in plague, both in Persia and in Mesopotamia, there happened some time before and again some time after the appearance of the severer forms of the disease, a series of milder cases which, so to speak, ushered in and ushered out the severest variety, or *pestis siderans*.

According to Dr. Hirsch, the first undoubted case of plague in Vetlanka was that of the woman Mawra Pisarewa, who went by boat from Vetlanka to Astrakhan *viâ* Vorpost, which faces Astrakhan on the opposite side of the river Volga. She visited a sick relation, a Russian Cossack soldier, who had just returned from the war in Armenia. It will be recollected that between Armenia and Mesopotamia communications were continual and uncontrolled. Dr. Hirsch thinks that in spite of official contradiction there is every reason to believe that the plague had visited both the armies of the Turks and the Russians in Armenia.

Even if the Cossack soldiers were themselves free from the plague, it is still possible that their clothes or effects may have at the seat of war become plague-infected. Many are of the opinion that plague-infected effects, including the booty and pillage of war, may long retain the poison, especially if the effects be packed up in tightly-closed boxes. Such packages, even after a long time, on being reopened would be likely to give forth the poison of plague-contagion. Dr. Sommerbrodt, one of the German Commissioners, relates two cases of plague distinctly traceable, in the opinion of the Commissioners, to infection from clothes.

The investigations relative to the weather, the nature of the soil, and the sanitary conditions of the dwellings, threw no light upon the question as to whether such exterior causes influence the course and character of the plague.

The German Commission openly declare that had the Russian Government been served by a medical staff of higher class than then existed in Astrakhan, the plague at Vetlanka would have been much less intense; and they further observe that the Russian executive fails to obtain from Astrakhan either reliable or early information and advice upon sanitary and medical matters.

The Central Russian Government, after receiving news of the outbreak, first sent out Dr. Koch to Astrakhan. He was presently seized with plague and carried off by the

disease on the 15th* of December. Next Dr. Morosow was sent, who, on the 28th of December, fell also a victim. Dr. Grigoriew followed, and became, on the 8th of January, the third medical victim. Lastly, Dr. Krassowski was sent, but when he arrived at Vetlanka plague had almost disappeared.

In Vetlanka, until the 18th of December no precautionary sanitary measures were taken. The inhabitants acted upon their own opinions, unaided and undirected by any experienced or recognised expert.

During the panic it was found impossible to get the plague-corpses buried, hence the bodies remained uninterred till the termination of the epidemic. Even then the dead were only thrown into pits and loosely covered up with earth to the extent of about a foot. Later, over these first interments, some lime was thrown and made firm by the addition of a layer of clay, the ground being raised to about the height of three feet above the corpses. It was still later before the pest-stricken houses were cleansed and disinfected.

Count Loris Melikof arrived towards the end of January, and issued a proclamation on the 2nd of February for the cleansing and scavenging of all streets in the towns and villages of the province. The burial-places were also inspected, and their condition improved. The sale of food was watched over by a sanitary executive. Means of disinfection were kept in readiness so as to be used when required.

For forty-two days after the last death from plague a strict sanitary cordon around the infected district was maintained. Fixed or permanent stations were established at intervals of every two miles and maintained by Cossack soldiers. Between these stations were patrols. It was given out by the Russian authorities that during the night extra intermediate patrols were placed. On both banks of the river Volga the cordon was enforced. During the period when the stream was frozen over, quarantine was carried out on the Volga by guard-posts on the ice, and later by guard-boats. Cordons were also established around the infected villages. Some of the special cordons were abolished on the 15th of March, whilst the general cordons were not removed till the end of April 1879.

Quarantine lasted at Samiani from the middle of January till the end of April 1879; at Sietlojar from the 29th of

* The dates are given in Old Style.

December 1878, till the 15th of March 1879; at Serepta* from the 27th of December 1878, till the end of March 1879. Here, as throughout this report, the dates are given in the old style.

The quarantine arrangements observed during the Commissioners' tour of inspection are thus described:

A. A reception-house for examining and disinfecting papers and passports.

B. At a distance of some fifty yards from A were two buildings to accommodate persons suspected of being infected by the plague.

C. An establishment for disinfecting clothes.

D. The lazaret or infirmary for plague patients.

E. The lazaret for those suffering from suspicious or contagious diseases.

F. The lazaret for those thought to have non-suspicious and non-contagious diseases.

Now A, the reception-house for examining papers and passports, was isolated by a hedge. In it were stationed the local army-surgeon, a police official, and some Cossacks. The doctor with a pair of tongs took up the passport into the disinfecting chamber containing a well-closed wooden box or case, having within it over a sand-bath a saucer holding a mixture of 3 parts of superoxide of manganese, 7½ parts of sulphuric acid, and 6 parts of chloride of sodium. The process of each disinfection was continued for an hour.

B. The two buildings destined to receive those who were suspected of plague were separated from each other by a space of two yards. The one building comprised a large common room for the admission of ordinary people, such as peasants and the like. The other building consisted of four small rooms, and was set apart for the service of people of distinction.

Native tents were kept in readiness to meet the exigency of any sudden increase of plague-suspected guests.

Outside these buildings a watch was kept by two attendants, termed the overseer and the guardian, and who wore suits consisting entirely of leather. The overseer through the opened window was presumed to watch the condition of a patient. The guardian brought the food into an ante-room, and, on his retiring, then the quarantined individual was allowed to carry into the inner apartment his own meal.

* Sarepta is a fortified town on the river Sarpa near its influx into the Volga, fifteen miles south of the Tzaritzin. Its population is given by Keith-Johnston as 4,500. It is the centre of numerous colonies of Moravians. It has a custom house, schools, and manufactures of cottons, silks, woollens, hosiery, and tobacco.—J. L. H.

Afterwards the guardian was required to remove, with a pair of tongs, the vessels and appliances used in eating to the disinfecting-room.

c. The building for the disinfection of clothes numbered two rooms, in one of which linen and other washable clothes were soaked for two hours in a solution of chloride of lime; whilst the unwashable clothing underwent disinfection in the same manner as described for the papers and passports.

The servants who attended to this department, and waited on those suspected to be plague-infected, wore leather suits covered with tar.

The washable linen and clothing belonging to those suspected of being plague-infected underwent twelve hours' disinfection, with forty-eight hours' subsequent airing. When, however, it was discovered that the washable clothes came positively from plague-stricken individuals, then the process of disinfection was repeated and continued twelve hours; with forty-eight hours' subsequent airing, *plus* twelve hours' further disinfection, and then the clothes were burnt!

In connection with the department for disinfecting clothes, there was a large open space to receive carriages and animals, in order that they might be subjected to quarantine.

The Russian executive does not look upon fish as being a cause of spreading the plague; but the places where the unloading of fish from the carts took place underwent disinfection with chloride of lime. The fish was then packed in other vehicles, or possibly in the same vehicles after these had been cleansed and disinfected. (?)

In Simiani the extra reserve tents were those of a Russian military ambulance, being very similar in character to the tents used in the Russian army.

All effects, clothing, paper, and copper money had to undergo twenty-four hours' disinfection. Carriages were covered over with stretched-out tarred sail-cloth, and then well smoked and disinfected under a capacious tent.

In Samiani the disinfecting mixture was composed of 6 parts of manganese, 9 parts of common cooking-salt, 7½ parts of concentrated sulphuric acid, and 7½ parts of tepid water.

Immediately after the removal of the cordon at Vetlanka, the civil and military authorities, after having valued the plague-visited houses, and the suspected plague-infected effects, burnt all these properties.

The Russian Government paid for burning houses and goods, 69,000 roubles—(a silver rouble is equal to 3*s.* 2*d.* in English money)—of which 50,000 was compensation for eighty-three houses, and 5,000 roubles for the work of pulling down the buildings. 14,000 roubles were set aside as compensation for the destruction of clothes.

A number of medical men, under the presidency of the military surgeon, Dr. Reutlinger, were stationed for some time in the infected locality after the cessation of the outbreak to supervise the measures of precaution adopted against any recurrence. The cordons were very strictly kept; but the German Commissioners were not informed, as had been reported, that persons trying to break through the cordon were shot by the executive.

The quarantine arrangements appear, however, to have been very primitive, and the stations were too small for the number of persons requiring admittance. They were, moreover, too near together. The quarantine laws, indeed, could not be rigorously enforced; and the internal arrangement of the quarantine stations is stated to have been so defective that many of the healthy inmates became victims to the epidemic.

The conclusions of the German Medical Commissioners are thus summed up :—

(1) Between the commencement of October 1878, and the close of January 1879, a malignant disease prevailed in the province of Astrakhan, visiting six districts, in one of which only an epidemic diffusion took place. In all, from 450 to 500 deaths occurred.

(2) The disease presented the distinctive characters of oriental plague.

(3) The origin of this attack of the plague is uncertain Plague is not indigenous to the province of Astrakhan; and it cannot be clearly proved that the disease was imported from Resht, in Persia, during the year 1877.

It would seem most probable that the outburst was due to importation of plague-infected articles from the Asiatic seat of the Russo-Turkish War.

This epidemic affords a further illustration of the difficulty, even after careful investigations, of discovering the direct origin of the plague.

(4) Owing to the imperfect and faulty reports of the nature of the disease in Vetlanka, the Russian Government could not avail itself of preventative or precautionary measures until the outburst of plague had attained its maximum intensity, and was already approaching towards its natural ter-

mination. Even then the Russian executive were only prevailed upon to take up the matter *after* foreign nations and neighbouring powers had stopped their traffic with this part of the Czar's dominions, and *after* foreign scientific experts had been dispatched by foreign powers to investigate the disease.

ENDEMIC PLAGUE IN INDIA.

By Surgeon-General C. R. FRANCIS, M.B., etc., etc.

[*Read: June 2nd*, 1880.]

Introduction.—The phenomena of nature are exhibited in India on a colossal scale. A cyclone sweeps over a tract of country, and in a few hours its teeming populations are houseless. A storm-wave, engulfing hundreds of smaller craft in its onward course, raises the lordlier vessels from their supposed haven of refuge on the bosom of the sacred Bhagiruttee,* and deposits them on its banks alongside. The heavens discharge hailstones of more than regulation cricket-ball size and weight; and rain descends, not in inches but in feet. Famines destroy the human race by untold millions; and disease frequently assumes the characters of a pestilence. Two of the severest scourges known to man, viz., cholera and the plague, find congenial homes in India. It is of the latter disease, especially as it exists in a part of the Himalayahs, that I propose now to give a brief sketch.

I was one of two medical officers of the Indian Army who were nominated in 1853 to investigate this plague; and Dr. Pearson my coadjutor and myself were thus engaged for two consecutive years; at the end of which time the former was permanently appointed as superintendent of the measures considered necessary for its repression, in addition to other duties affecting the welfare of the people generally, *e.g.*, in promoting vaccination. Dr. Pearson, who has recently retired, was employed on this duty for upwards of twenty-five years.

The result of our joint proceedings was recorded, by permission of the Medical Board, in the *Indian Annals of Medical Science*, vol. i, 1854; but I am not aware that, with the exception of a valuable paper read by Surgeon-General John Murray, on the 10th April 1878, before a meeting of this Society, and recorded in the second part of the fourth volume of its *Transactions*, any separate account of this endemic plague

* Synonymous with the Hooghly river off Calcutta.

of India has ever appeared in any of the professional periodicals published in this country.

Physical Geography and Geology.—Before proceeding further it may be well to refer briefly to the physical geography and geology of the country in which this scourge maintains its ground, and to the people that inhabit it. On looking at a map of India, we find, situated in the great Himalayan chain, in 29 deg. 45 min. N.L. and 79 deg. 45 min. E.L., the district known as Kumaon, with, extending in a N.W. direction into 30 deg. N.L. and 78 deg. 40 min. E.L., the attached sub-district of Gurhwal.* Kumaon and Gurhwal cover a space of about 11,000 square miles, half of which only is fit for cultivation. Of this half, three-fifths are always covered with snow; one-fifth *is* cultivated; and the remainder *is not*. The population, especially in Gurhwal, where, in former years, the people endured great tyranny from their rulers the Ghoorkhas—many being annually carried off under the operation of a slave tax—has now become sparse and scattered. This tract of country, the only part of the Himalayahs where *mahamurree* has ever appeared—low forms of fever are common enough throughout the entire mountainous chain—is bounded, on the north by the watershed line of the main range and the Thibetan frontier; by the plains of India on the south; by the Alaknunda, one of the two main streams (that unite to form the Ganges) dividing it from the rest of the mountainous districts, on the west; and, on the east, by the great river Sardah (or Kalee) that separates it from the adjoining kingdom of Nepaul. The direction of the several mountain chains, including the main range and the inferior peaks, is from about N.W. to S.E. There is a general parallelism throughout, and the strike of the several strata is pretty much in the same direction. At the immediate base of these magnificent hills (a distance of from 80 to 90 miles intervening between this point and the line of maximum elevation) lies an equally magnificent forest, from 10 to 15 miles in breadth, which would require the genius and pen of a Humboldt justly to describe. No natural rivers are found in this region, and, until the officers of the British Government provided a more or less permanent supply of water through the medium of canals, there was no cultivation. Torrents, which rush impetuously down from the mountains, are met with in the rainy season; but the hill *rivers*, on reaching the commencement of the forest, are

* From Gurh, a fortress, there being so many fastnesses capable of being so used.

immediately absorbed into it, and, passing onwards underneath the surface to the plains, reappear in the moist tract which—of almost equal breadth with its dry neighbour—is remarkable for its swampy character, especially marked during the rains. This tract, covered with tall reeds and grasses, and intersected by sluggish streams, often doubling back upon themselves—a mixed swamp and prairie—is known as the *Turaee** proper, in contradistinction to the dry forest, which is called the *Bhabur*.† The latter, whose surface consists of a few feet of earth resting on a dry bed of shingle, boulders and sand, indicating the bed of a former sea,—with a splendid climate in the cold months, but *intensely hot* in the hot season,—without, comparatively any malarious influences,—has no permanent population; whilst the former, essentially alluvial, without a trace of rock, fragmentary or otherwise, and *full of malaria*, can boast only of a few wretched villages, inhabited by a miserably attenuated population.

Elevation.—The average altitude of the mountain ridges is about 7,000 feet above the level of the sea. Some of the snow-clad peaks attain an elevation of 23,000 feet and upwards. Nunda Devi, the highest in the range in this part of the Himalayas, is more than 26,000 feet high.

Geology.—The mountains generally are composed of rocks of igneous origin, with, reckoning from the south, argillaceous schists, grit, and limestone. The lines of igneous action follow, for the most part, the direction of the strike. Proceeding northwards, we come upon a tract of some breadth, consisting mainly of mica schist, with a burst of granite along its central part. Still further north we find slates, limestones, quartzites, these also being traversed by lines of igneous action. In these lines iron and copper have been discovered. The eruptive rocks consist largely of greenstone, associated with black basalt, passing into schists and conglomerates. Breccias, known as ash or "volcanic grit", are not uncommon. Here, too, steatite, and an occasional magnesian bed are found. In all this part of the mountains there are no fossils. To the north of the great snowy peaks, at 14,000 feet, we come upon palæozoic strata (their lowest beds being of silurian age), with limestones, argillaceous shales, followed by oolitic beds, and Oxford clay. Characteristic fossils—from silu-

* From *turee*, Persian for moisture, or from *tale*, Sanscrit for down below, foot of.

† Not known why so called. Supposed by some, though incorrectly, to be taken from a kind of grass called Bhawur; but this does not grow in the Bhabur; it is found only in the lower hills.

rian to tertiary formations—are here found, throughout. On the Thibetan plateau (120 miles in length, from fifty to sixty miles in breadth, and from 14,000 to 18,000 feet above the sea's level)—a great tertiary deposit, on which are found boulders, gravel, clay, and mud (the ancient bed of a sea)—we meet with bones of the elephant, rhinoceros, horse, hippotherium, and several ruminants. The outer ranges of the smaller hills, running parallel with the higher, and consisting mostly of sandstone and conglomerate, are—to the west of Hurdwar, where they are known as the Sewalik range—famous for the palæontological discoveries of Colonel Cantley and Dr. Falconer.

Ethnology.—Whatever be the exact ethnological position of these several hill tribes—it has been affirmed that they are of mixed Thibetan and Hindoo birth—it is evident that the Kumaonees and Gurhwalees, who call themselves Khas-yas, a race of Kshatriyas, who are widely distributed throughout the Indian Himalayahs, are essentially of Hindoo origin They claim to be descended from the Rajpoots, only part of the ar removed from the finest specimens of appeared—low formitat, so to speak, is Rajpootana, in the out the entire mountai.

by the watershed line ot the d Gurhwal are, for the most frontier; by the plains of Indiastes being the artizans of nunda, one of the two main streahed to their homes, offers Ganges) dividing it from the rest oads, away from them, tricts, on the west; and, on the east, convey a load from Sardah (or Kalee) that separates it frorather than earn kingdom of Nepaul. The direction of the sertaking what chains, including the main range and the infeht! In this from about N.W. to S.E. There is a generathe Kumaothroughout, and the strike of the several stratalive near much in the same direction. At the immediatAlmorah, these magnificent hills (a distance of from 80 to 9isitors' intervening between this point and the line of mai from elevation) lies an equally magnificent forest, from 10 tes- miles in breadth, which would require the genius and pel of a Humboldt justly to describe. No natural rivers an found in this region, and, until the officers of the Bri' Government provided a more or less permanent supplee water through the medium of canals, there was no cultpe) tion. Torrents, which rush impetuously down from ta- mountains, are met with in the rainy season; but the hr. *rivers,* on reaching the commencement of the forest, an

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surroundings are less insanitary; though, in many cases, there is but little difference in this last respect between them.

There is one essential distinction in the clothing of these hill people. The Kumaonee's garments are of cotton, the Gurhwalee's being of wool; and as these are often worn threadbare, this fact, added to want of individual cleanliness, and the absorbing capacity of the material, gives strength to the conclusion that personal filth invites the disease. Between the mountainous regions proper, and the great Thibetan plateau, is situated the country called Bhot, where the climate is very inhospitable, and the crops that are grown there (consisting of amaranth, barley, buckwheat, and wheat) are, consequently, very poor and uncertain. Here are located the Bhotiyah villages, inhabited by a hill tribe who, also Hindoos, closely resemble the people of Thibet. The Bhotiyahs (such is their name), short in stature, but of a powerful physique, are the carriers of the hills (for this privilege they pay tribute to the Thibetan Government); taking down to the plains of India the produce of Thibet (or, as it is locally called, Hoondes, or snow country), consisting mainly of borax and salt (the greater part of which is consumed in the hills), wool (pashm or shawl wool), woollen cloths, and shawls of superior quality, silks, ponies, cows' tails, etc., and bringing back cloths of various kinds, sugar, hardware, tobacco, spices, and miscellaneous commodities. They also carry into Hoondes, which has no agricultural produce, the grain that is largely grown in the villages to the immediate south of those inhabited by themselves. These goods are conveyed on the backs of sheep, or, where the loads are heavier, of jubboos* and goats, guarded by large dogs, whose watchful fidelity, as evinced by their incessant barking through the livelong night, greatly disturbs the rest of the weary and unappreciating traveller in his tent.

The Bhotiyah villages are located north of the great peaks, at elevations of from 7,000 to 12,000 feet above the level of the sea; and, so far as I know, the plague has never appeared in them, nor amongst the Bhotiyahs in their transit through the hills, any more than it has (of late years, at any rate) amongst the pilgrims from the plains. These last annually visit the several shrines established at the origins of the sacred rivers; as at Jumnotri, the origin of the Jumna, and Gungotri, the origin of the Ganges, etc. They sometimes assemble in large numbers at these shrines; and, from wear and tear of travel, are obviously predisposed to the invasions of epidemic disease.

* The jubboo is a cross between the yak of Thibet and the cow.

Outbreak of Mahamurree at Kedarnath.—It is probable that it was at one of these large gatherings where the recorded outbreak (at Kedarnath, in 1823) of mahamurree occurred; though the natives themselves attribute it to Divine vengeance, which thus manifested itself as a punishment for improper administration, on the part of the high priest, of religious rites. The combination of insanitary conditions doubtless existed in full force at Kedarnath and at such gatherings in those, as also in later, days; and altitude would not avail against them. We know how those conditions have operated, in more recent times, at fairs and pilgrim resorts in the plains.

Local Nomenclature of the Plague.—The local names by which the plague in the Himalayahs is known, are mahamurree, from *Maha*, a Sanscrit word, signifying great, and *Murree*, the meaning of which is not very clear. It may be derived from the Sanscrit *Mara*, dead; and thus the word would imply *great death*. It is also called *Pootkia* rog, and *Gola* rog. Of the derivation of the former I am not aware, unless it be from the plant, so called, that is used for treating the malady; or it may be *vice versâ*, the plant being so named from its use. It may be connected with the bursting of the buboes, etc.; the Sanscrit verb, *phutna*, meaning, to burst. Gola is of Sanscrit origin, signifying ball (referring to the glandular swellings). Rog is Sanscrit for disease.

There is no reason why mahamurree should not have existed at a much earlier date than 1823. Doubtless, with the prolonged sojourn of a people (whose filthy habits are probably unsurpassed by those of any nation in the world) on the same soil, which would thus, year after year, be made a very hot bed of pollution, the disease became intensified, and spread.

Advance of the Plague Southwards.—From Kedarnath it gradually extended itself downwards. For many years, however, it was limited to certain northern portions of the district, known, locally, as Pergunnahs Nagpoor and Budhan. Eventually, it descended to within fifteen miles of Almorah, the modern capital of Kumaon.

It would appear that mahamurree first attracted official attention in February 1836; but, beyond a "report", nothing was done. Again, during the rains in 1849, the magisterial authority in Gurhwal brought the existence of the disease to Government notice. So severe had it now become, that the inhabitants were fleeing from their homes in terror, and the annual revenue was not forthcoming. In April of the following year, the medical administrative

officer of the Circle visited, by order of Government, some of the affected spots; and, though he did not then realise the identity of mahamurree with plague, Dr. Renny saw that a fever of unusual severity had established itself as a plague spot in the land, and that a searching enquiry, in view to, if possible, its removal, was urgently called for. Thus, it came about that Dr. Pearson and myself were appointed, in 1853, to undertake such an enquiry on an organised plan, some preliminary observations having been made, in October 1851, by the former, in conjunction with the (then called) sub-assistant-surgeon of Almorah, Baboo Sree Nath Mookerjee; supplemented by yet other observation, of a month's duration, by Dr. Pearson and myself. *It was time for an organised enquiry.* In 1834, an outbreak of mahamurree had caused 633 deaths in Gurhwal—a large mortality in so thinly populated a district. In 1849-50, 111 victims had succumbed in nine villages. The maximum death-rate was estimated at 88, and the minimum at 25, per cent! The worst outbreak occurred in 1852. The disease burst forth in that year almost simultaneously (though contagion, as a factor, was distinctly traceable in two or three) in twenty villages, where formerly one only had been visited. The increasing intensity of the disease, added to its southward march, inspired the authorities with justifiable alarm.

Having thus given a slight outline of the former history of mahamurree, I proceed (*a*) to describe the disease as it has been seen by various observers and by myself: (*b*) to suggest what may be the probable cause of it: and, (*c*) finally, to offer a few remarks as to the most suitable remedial measures.

Symptoms.—Mahamurree, usually ushered in with shivering, is essentially a fever of a most virulent and fatal type, marked by rapid and extreme prostration, great præcordial oppression, and associated, in many cases, with glandular swellings, the suppuration of which is regarded as critical. A white chalky, laterally fissured, but unswollen, tongue, occasionally resembling the strawberry tongue of scarlatina; with headache, accompanied not infrequently by passive delirium and imaginary noises within the brain; a peculiar mixture of muddiness and lustre in the eyes; and constipation (though, now and then, there is diarrhœa) are marked features in the disease. There is sometimes acute pain in the loins, with scanty and high-coloured urine. Strangury is more rarely noticed. The character of the pulse varies. In one case it would be 90 and full, though

very compressible: in another so weak and rapid, that it was difficult to count it. Where perspiration supervened, either naturally or as the result of remedies, it was considered highly favourable. I have known the perspiration, in a case of recovery, literally pour from the surface of the body—the patient being, when in health, a hale, hearty man. Vomiting was not a common symptom, but the natives thought well of it when it occurred. A wish for bitter things would be often expressed. The course of an attack would sometimes be startlingly brief. It was not at all uncommon for an individual to be suffering in the morning from nothing, apparently, but malaise; yet, in twenty-four hours, that sufferer would be a corpse, so overpowering was the depressing effect of the poison upon the nervous system. The greater number of the cases proved fatal on the third day.

Glandular Swellings.—Where there were glandular swellings, these usually appeared on the fourth day (but sometimes much later) either in the inguinal, or femoral, or auxiliary, or cervical region. Occasionally, they were seen immediately below the ear. Except in the rapidly fatal cases, the fifth was the critical day. If the patient tided over this day, he was probably safe; though there was anxiety till the eighth. When glandular swellings appeared the alarm became greater, and their suppuration was eagerly watched for. Their recession was a bad sign. The natives would push a large heated needle through these swellings, to promote the formation of pus. It may be mentioned here that this, and the internal exhibition of the milky juice of the *pootkia* (a species of *euphorbium*), to cause evacuations from the bowels, which were usually costive, constituted all the native treatment of mahamurree.

Petechiæ.—It does not appear, from the writings of the several observers of the disease, that petechiæ were ever seen. I myself did not notice them in any one of the ten cases gathered from the fifty villages that Dr. Pearson and I saw together, or separately. Dr. Pearson, whose experience of mahamurree has extended over more than a quarter of a century, informs me, in a letter recently received, that he has occasionally met with petechiæ. The dusky skin of the natives of these hills would make the detection of spots somewhat difficult, especially where a layer or so of dirt was super-imposed. Still, as they have been looked for by several observers, and seen, so far as I know, only by one, it seems evident that they are not very common.

Hæmorrhages.—So with hæmorrhages. These are met

with, if at all, in the quickly fatal cases; but hæmorrhage is not an ordinary accompaniment. When seen it is in the form of hæmoptysis.

What is Mahamurree?—In answer to the question "What is mahamurree?" the most experienced observers have no hestitation in saying "Plague"; a genuine specimen of the *pestis septica*, or Egyptian (or Levantine) plague. I believe that Dr. Murchison correctly defined it when he wrote: "Plague is perhaps the typhus of warm climates.....differing only in intensity from the effects of climate and other collateral circumstances." He adds, "this disease closely resembles, if it be not identical with, bubonic plague;" and Pettenkoffer is of the same opinion.

Dr. Morehead, in his valuable *Clinical Researches on Disease in India*, thinks that mahamurree and Pali plague—a disease to which I shall refer later on—are adynamic remittent fevers of suspected infectious character. Malaria, he says, may be the original agent; but let there be overcrowding and you get the poison of typhus in addition. However well this definition may apply to some forms of fever that have appeared in India, I venture to affirm that it is not applicable to mahamurree. Neither intermission nor remission have been observed in the course of an attack. Sulphate of quinine has had no appreciable effect upon one, as in fevers of malarious origin; and the general expression of the countenance has always been pathognomonic. Moreover, the chief distinguishing features of plague are present, viz., the rapid course of the disease, the glandular swellings, with occasional hæmorrhage; and the excessive mortality, with the absence, often, of any prominent symptoms.

Post-mortem Examinations.—As before stated, Dr. Pearson and I, when working together, placed on record the particulars of ten cases (amongst which there were only two recoveries), which I refrain from repeating here, as the symptoms which they severally embodied have been already enumerated. We made four *post mortem* examinations; but beyond general and excessive congestion, which existed in every organ in the body, from the brain (in which it was most prominent) downwards, we found nothing; there was no special lesion. In the lungs, however, of the rat which we examined, there were on the surface what looked like carbonaceous patches—islands of hæmorrhagic effusion.

Cause of Mahamurree.—We come now to the questions "How does mahamurree originate? and how is it propagated?" I am afraid that we have no better answer to the first question than we had thirty years ago. We now

know indeed, as we presumed then, that insanitation fosters the disease and doubtless invites outbreaks: for a relaxation of hygienic regulations (partly as the result of the mutiny of 1857, and partly, it must be added, in consequence of the chief civil authorities in Kumaon not believing in and therefore not rigidly enforcing them) always has been followed by the reappearance of the disease in as virulent a form as ever. From 1854 to 1857, during which period, owing to the energy of Mr. (now Sir John) Strachey in Gurhwal, sanitary progress was there most vigorous and effective, the plague was comparatively quiescent: but, in 1859, and again in 1860, it visited the northern Pergunnahs in Kumaon with great severity; and, in these years, 1,000 persons died from the disease. Again, in 1876-77 there occurred 291 cases, of which 277 were fatal—a death rate of about 95 per cent.! (The official returns show that 3,600 deaths from mahamurree have occurred since its first appearance in 1823.) Until hygienic measures were adopted, the general uncleanliness of the people in their persons and *entourage* was incredible. A small stone dwelling (built upon a surface 13 feet square) consisting of two rooms, each about 5 feet high, one above another,—the upper chimneyless and practically windowless; tenanted by the entire family of often more than half-a-dozen in number, and by the huge baskets containing the family grain; the lower compartment (a wooden floor, full of cracks serving as media for the effluvium from below, dividing the two) being occupied by the family herds, consisting of cows, goats, and pigs; a row of such dwellings (sometimes they are single, or double, spread over an irregular surface) similarly tenanted, and flanked at either extremity by the ancestral heap of manure from which streamlets of liquid filth were flowing in different directions; the cottages covered with cucurbitaceous creepers, as, cucumbers, pumpkins, melons, and the like; a small forest of hemp, some 8 or 10 feet high, luxuriating in the immediate neighbourhood of the village; a growth of underwood, including nettles, etc., between the two, and more or less surrounding the latter; and unwashed Paterfamilias, seated in front of his fig-tree, having submitted his head to be divested by a faithful spouse of the light infantry skirmishing in his unkempt hair! Conceive such a village situated towards the base of a mountainous slope, well within the range of whatever noxious influences may emanate from the valley below; located where there would be the veriest minimum of ventilation; and we cannot be surprised if, when sickness

does come, it should run rampant. The atmosphere and peculiar smell in these locations must be *encountered* to be appreciated. They are *sui generis*, and are very suggestive of disease.

Caprice of Mahamurree.—The caprice, as it is the fashion to call it, of mahamurree is, like the caprice of cholera, sometimes displayed in a singular manner. There may be two villages placed under apparently precisely similar circumstances; and the one shall be attacked, the other not. I have two such in my recollection. Both situated on the same mountain steep and imbedded in the same jungle, with the same N.N.E. aspect, were only five hundred yards apart; and yet, at every visitation of mahamurree, whilst one has always escaped, the other—Kunowlee—has not. Doubtless there is some condition, the nature of which has not yet been fathomed, that secures the exemption.

Death of Rats.—One very singular circumstance is the almost invariable death of the rats* of the village, which occurs on the eve of an outbreak of mahamurree. One would suppose that, as these animals only die in a particular way, and in some numbers at such times, the natives would at once take warning and vacate their villages; but no—they linger on until one of themselves falls a victim, and then occurs a universal exodus into the jungle, all obligations from ties of kindred being lost sight of in their personal terror; though we witnessed some notable exceptions to this general display of selfishness. Beyond letting the village remain uninhabited, for a time, the people would take no steps to improve its healthiness. At the end of a few months they would go back and live in it as if nothing had happened.

Mode of a Rat's Death.—The manner of a rat's death was remarkable: the animal would emerge from its hole on to the floor, stagger, perform an involuntary gyration or two, bring up blood, and die. More commonly the rats were found in the morning lying dead near the sleepers. On more than one occasion a rag has been found between the clenched teeth of the rat. I am not aware that any of

* A similar preliminary mortality amongst the rats occurs in Yunnan, in Western China, where the disease, evidently plague, has been endemic for some years past, especially since the occurrence of the massacres which accompanied the outbreak and suppression of the Mahometan rebellion in that province; the surrounding provinces remaining entirely free from it. Here the plague is invariably preceded by a great mortality among the rats and other animals living in drains and in holes in the ground. As soon as this premonitory warning is recognised, the people .....purify their houses, abstain from pork, and in some cases fly from the threatened district.

the professional observers ever *saw* a rat die, and as thus stated; but, as the same statement was universally made by the natives, there is no reason to discredit it. And we saw rats dead.

Examination of the Grain.—An idea having prevailed in some quarters that diseased grain (that in common use is the *Eleusine Coracana* known locally as *Mundooa*) might be a factor in the generation of mahamurree, we carefully examined it, as stowed away in the baskets, from top to bottom; but it was good throughout, without any trace of fungus or any disease whatever. The pultaceous mass of food (consisting of grain) found in the rat's stomach seemed particularly wholesome. And, on their return home from the jungles, the survivors, if they find any grain left by the bears—who generally, however, make a pretty good clearance—entertain no scruples about using it again. Indeed, they sometimes take it into the jungle with them. Doubtless it is diseased sometimes; and the people, who cannot afford to throw it away, do eat it in that state: but I very much doubt its ever lighting up mahamurree: and, if we admit it as a cause of the disease, it should be in universal operation.

Death of other Animals.—From the alleged fact of snakes and jackals being found dead, as well as rats, the diseased grain theory found supporters, who argued that, as the rat eat the grain, and the snake eat the rat, and the jackal eat the snake, the grain, somewhat on the principle of the house that Jack built, would be the original offender. According to this theory the cats ought especially to succumb; but none, I believe, die at these times more than at others. Those that we saw were, in common with the dogs of the villages, somewhat thin. Disease amongst animals on the appearance of a pestilence is, of course, no new fact. On the contrary, it has been noticed since the time of Livy's writings—the animals suffering first in the order of sequence. Arejula states that, on an outbreak of yellow fever at Cadiz in 1800, the dogs died first, then the cats, then the horses, then the poultry; and, lastly, canaries, from the bills of which, in some cases, blood issued. I may mention here that Dr. Pearson, in his long experience, saw no connection between mahamurree and the disorders of cattle. The cattle suffered occasionally from foot and mouth disease, but not especially in mahamurree years. During the outbreaks of this disease, and before, they, as a rule, remained healthy, as did the sheep and the goats. Dr. Watson (who succeeded Dr. Pearson) attributes their

immunity from the plague to the fact of their eating only grass and no grain. He gives, in support of the grain theory, the case of rats, in the neighbourhood of a water-mill, beginning to die only when some grain had been taken from a village to be ground. With the exception of rats, and much more rarely, snakes,* the death of animals in connection with mahamurree was not a marked feature. If one animal, eating another plague-infected, should die, how much more the bears who, it is alleged, consume the bodies of the villagers that have died of mahamurree? Certain it is that bears are often carnivorously disposed; and that they were observed in greater numbers than usual in outbreaks of this plague. I myself saw, one moonlight night, what appeared to be a small family of bears pass the bamboo scaffolding on which I was seated—they were too far for a shot—and the plague-stricken corpses rapidly disappeared. No dead bears were ever seen.

Escape of Pilgrims.—A colour has been given to the diseased-grain theory in consequence of the pilgrims, who annually visit the hill shrines in large numbers, eating other than the village grain (their own, which they carry with them, whilst it lasts) and escaping. But, in the northern villages (those immediately below the snows, where grain is largely grown for consumption in Hoondes, as already mentioned) there is a considerable traffic in grain with the pilgrims, who here replenish their stores. Dr. Watson says that they do not eat the common grain of the hills, in which a poisonous fungus is apt to form, but wheat, barley, rice, and pulse, in which it does not. Dr. Watson speaks of two pilgrims dying from mahamurree; but I do not think that he saw them. I have written for the favour of being furnished with particulars. The immunity of the pilgrims is probably attributable very much to the circumstance of their holding, with the exception just mentioned, little or no intercourse with the hill villagers. The roads to the shrines, as a rule, *skirt* the villages; they do not frequently pass through them. An infected village would, of course, be studiously avoided. The pilgrims from the plains, whence these worshippers come, are Hindoos, who think much of themselves, and look down upon their Hindoo brethren of the mountains. Of even a Brahmin amongst them they would sneering say, "Oh! he is only a *hill* Brahmin." Hence the communication with each other is, except where absolutely necessary, very limited. Moreover, the body-clothing of the

* Possibly more dead snakes might have been found, if looked for, in their holes.

pilgrims is made of cotton. During their progress in the hills (short of the snow) the temperature is rarely below 75 deg., and it is a curious fact that, in the upper regions, where there might be danger, as Okermuth, on the Kedarnath road, and Joshimuth, on that to Budrinath, the two* pergunnahs, through which the pilgrims pass, are uniformly free from mahamurree, though it may be raging in neighbouring pergunnahs†.

To return to the question, "What is the essential cause of mahamurree?" I trust I may be pardoned for quoting here the views of Dr. Murchison on the origin of disease, appealing as they do to our common sense. He says, "The poisons of all diseases must have originated at one time or another independently of a pre-existing case. Contagion necessarily implies the presence of two individuals, the giver and the receiver of the morbid germ. It is self-evident, then, that in the first sufferer from any disease its origin must have been *de novo*; and there is no reason why the unknown causes of the first case may not operate at the present day. The history of medicine, moreover, shows that new contagious diseases have from time to time appeared, while old ones have died out." Murchison adds, speaking of typhus, "Although in a large proportion of cases, especially during epidemics, the specific poison is derived from persons previously infected (contagion), it is, I believe, equally true that it may be generated independently." He then describes the conditions under which typhus may originate, the conditions which, in the most essential points, are rife in Gurhwal, viz., "overcrowding of squalid human beings, and deficient ventilation; in other words, the poison is generated by the concentration of the exhalations from living beings, whose bodies and clothing are in a state of great filth." "The great predisposing cause", Murchison continues, "is defective nutrition." Now, the aspect of a Gurhwallee does not convey the idea of want of stamina, and yet he has but little. His food, he will tell you, consists of *Mundooa ke rotee awr sheeshna ke sag, i.e.*, of unleavened cakes of the eleusine above referred to, and boiled nettles for vegetables. The nitrogenous element in this combination is not abundant.

Climatic.—Mahamurree, flourishing in a plague latitude, is a temperate climate disease, occurring for the most part, says Dr. Pearson, during the hot and rainy season; though, under favourable circumstances, it has, like other hot-weather diseases, appeared (and with virulence) in the

* Dasoli and Painkhunda. † See Dr. Watson's report.

cold weather: Its usual habit is to break out towards the close of the rainy season, and to continue, often, till the end of December, when it remains quiescent. It may again show itself in March or April, and continue till May, when it abates, and, for the time, disappears. Good asserts, and Sir G. Blane supports him, that, in Europe and Africa, the plague is destroyed or suspended between 60 deg. and 80 deg. Copland gives lower figures, viz., from 35 deg. to 75 deg. But mahamurree has appeared within a considerable range of temperature. I have seen it with the thermometer, in my tent, varying from 83 deg. to 95 deg., rising, in a grass hut, to 105 deg. But the atmosphere was more or less damp. It is very probable that, with a perfectly dry air, and the thermometer at 75 deg., or above it, the poison would be inert. Extremes of temperature, combined with moisture, are, as is well known, not unfavourable to the development of disease. But dry cold is bracing, and the healthiest season of the year in India is the hottest, when the dry hot winds are blowing their hardest. Kumaon and Gurhwal, as already stated, are in plague latitudes, as also are the plains of Upper India; but the native inhabitants of the latter escape mahamurree, it is alleged, in consequence of the great heat. Their personal cleanliness, and a better sanitary condition (though in many places there is room for improvement in this respect), doubtless conduce greatly to their immunity.

Poison in the Soil.—I strongly incline to the belief that the poison of mahamurree exists, in great force, in the soil, which becomes at every visitation more and more impregnated with it, owing to the filthy habits of the people. They contribute to its accumulation in every possible way. The very plague corpses are committed—those that are meddled with at all—to the earth, which is nothing but a rocky sub-soil, and most unsuitable for such interment; and they excavate it to a depth of not more than two feet, in immediate proximity moreover to their homes! A far better plan would be cremation (which might be adopted with cholera and small-pox cases also) on the banks of a stream leading (as so many of them do) to a sacred river. The death of rats and snakes favours the view of the poison emanating from the soil, but whether this be associated with electrical or volcanic changes in the rocks, or earthquakes, or the development of sulphuretted hydrogeu, we do not, as yet, know. Dr. Planck, the Sanitary Commissioner of the N.W. Provinces, who was deputed to investigate, in conjunction with Dr. Pearson, the outbreak of mahamurree

in 1876, believes that the rats die of suffocation. He comments upon the fact of these rats being unlike the large animal of Norway origin that frequents our sewers. They are grey and of a more delicate constitution. He discredits the diseased grain theory, as the rats are not found dead in their holes. I am not aware, however, whether these have ever been thoroughly searched.

Contagion.—The infecting power of mahamurree is undoubted: indeed, it has been spoken of as "intensely communicable". Amongst the numerous instances, illustrative of this truth, I may mention the following. At a village called Bintola, in November 1876, the child of the headman sickened with mahamurree, and died on the third day. This was the first case. Two other children in the same family also sickened shortly afterwards, and died. The unfortunate father owned two contiguous houses—his homestead; and his family consisted of fourteen souls. During November and December thirteen succumbed to the disease, which, in each case, set in with similar symptoms; shivering, intense fever, with delirium, followed by insensibility and death, which occurred on the third or fourth day. In the three houses adjoining those belonging to the headman lived three families, consisting likewise of fourteen persons. The women of these families helped to move the children of the headman. Soon, the disease, attacking them, carried off twelve of *their* number; passed on to the rest of the village and, in the two months, destroyed, out of thirty-one attacked, twenty-seven lives, including those in the huts to which the stricken ones had fled. Into another village—Balt—went a woman from her own (which was uninfected) to nurse the children of the headman *there*. She caught the disease, returned home and carried it with her; for almost immediately on her arrival mahamurree broke out.

Immunity of Europeans.—It is remarkable that no European has ever been attacked by mahamurree; yet, in the discharge of their duty, several medical officers have been exposed to the contagion. Dr. Watson, who succeeded Dr. Pearson, maintains that, if the doctor visits a patient in his house, or hut, on a cold damp evening, after sunset, and sits in it for a minute or two, and feels the pulse, he is almost sure to contract the disease, which will appear within twenty-four hours. A professional examination may be made with impunity, adds Dr. Watson, if the sun be shining and the air warm. Doubtless, this view is correct. Dr. Pearson and myself were careful to keep to windward of the patient, and to wash our hands (which we kept free

from abrasions) after contact with him; and we made a point of oiling them when performing the *post mortem* examinations. But Europeans, when travelling in these hills, are not under the conditions that predispose to an attack of mahamurree; and, with ordinary precautions, would be very unlikely to catch it.

Pali Plague.—The Himalayahs are not the only place in India where plague has appeared. It has raged with great virulence, though more temporarily, in the West of India, in the Bombay Presidency. Its first advent there was, it is believed, in May 1815, in Guzerat, Kuttywar, and Cutch, after three years of severe famine. It reappeared, the following January, February, and March, in the same locality, whence it migrated to Scinde. It seems to have remained in these parts till 1819, when it disappeared; and we hear no more of the disease till July 1836, when it broke out with violence at a town called Pali, in Marwar, whence the name by which it has subsequently been known —*Pali Plague.* Although not altogether a new disease, it first attracted official attention in 1836, the very year in which mahamurree was first brought to the notice of Government. It was remarked by a Gooroo (or priest)— "This seems to be a new disease, but it was known to the past generation;" a statement which points to the outbreak of 1815, extending over four years, till 1819. From Pali the disease radiated within a circuit of thirty miles, extending in October even as far as Joudhpoor. Under the energetic supervision of Major Dixon, then superintendent of Ajmere the capital of Rajpootana, quarantine was established; and the plague did not reach any other states in the province, the intervening hilly country of Mhairwarah helping to exclude them. One town, however, viz., Dewar, was affected, owing to infringement of the quarantine regulations. Interested traders persuaded the inhabitants to conduct porters carrying the bales of merchandise into Dewar, where the disease immediately afterwards broke out; passing over, in March (1837), to Deogurh in Meywar, in which district thirty-two villages in all were attacked. With the setting in of the hot season, in that same year, the plague declined, and has not since been heard of, as such, in that part of India. Medical officers, one and all, unhesitatingly called it infective plague. Whilst this plague was raging in Rajpootana, in Jilwara common remittent fever was very prevalent: in the intermediate space occupied by Jeypoor the intermittent of the country was rife; whilst, in Rohilcund—the district ex-

tending to the foot of the hills where mahamurree was doing its work—a deadly infecting epidemic resembling yellow fever was decimating the country. The concurrent existence of these several epidemics seems to show that the general meteorological (?) conditions were such as to call into action the operation of influences peculiar to each locality: malarious fevers in one, yellow fever in another, typhus in a third, and plague in a fourth. As before mentioned, no doubt was felt as to the nature of the Pali disease. An attack would be ushered in by shivering, with hot skin, great debility, universal tremor—so marked a symptom in mahamurree; great thirst, a white-coated tongue, fiery red papillæ showing through the coating in some cases at the edge; insensibility on the second day, followed by an early death. No petechiæ were seen, but glandular swellings were common. The mortality was usually 50 per cent., rising as high as 75. Convalescence, in the recovered cases, was very slow. The treatment adopted by the natives was even more simple than that for mahamurree. It merely consisted of hot stimulating fomentations to the glandular swellings. Said a barber-surgeon to one of the medical enquirers: "I never meddle with patients till after the fifth day."

Origin of Pali Plague.—Once started, the Pali plague was highly contagious: but the question was, HOW was it started? Was it imported? Some believed that it came from Egypt: but if so, it surely should have appeared first in the line of direct communication and traffic, as Bombay? but it was never seen in Bombay at all! The influence of decaying vegetation must be excluded, as there was none: neither was there any pestilential air from neighbouring jheels; which did not exist. Moreover, the fever was not of the kind that is generated by these causes. The soil was everywhere sandy and dry; and the towns were either built on such soil, or on the rocky bank of a running stream. It is far more probable that the Pali plague originated, like mahamurree, from animal poison, the conditions under which the people of Kuttywar, etc., lived, being pretty much the same as those affecting their fellow sufferers in the hills.

Sanitary.—The people were filthy beyond conception. They wore a large quantity of clothing, which was seldom changed. It was sometimes even allowed to rot on the person. As in the hills, the family herds lived under the same roof as their masters; and the dwellings, as also the bazaars and alleys, were always over-crowded.

Commenced amongst the Chippahs.—We find that the Pali plague invariably appeared amongst one class of people, viz., the Chippahs, or cloth-printers of Pali: that it then attacked others, who fled and formed fresh foci of contagion. The Chippahs principally had dealings with the bunnyahs, or intermediate small traders, who thus became the carriers of the poison. Why should the cotton-printers be the first to suffer? Because, it is urged, they imported the merchandise from Surat, on the coast; because they unpacked it; and because it consisted of goods brought, in part, from plague countries; whence it would, therefore, contain the fomites of contagion. Surat has commercial intercourse with Egypt, the Red Sea, and the Persian Gulf. In Kuttywar, as at Pali, these importers of cotton goods also *worked* in cotton—they made cotton cloths. From whatever source the poison came, the avocation of the Chippahs invited its advent amongst themselves, raw cotton affording a favourable nidus for its abode, just as the woollen dress of the Gurhwallee more readily gave a lodging to the poison of mahamurree, than did the cotton garment of the Kumaonee. Cotton raw, and cotton made up, are of course two very different things. The theory of packed goods transporting the poison, and disseminating it when those goods reached their destination and were unpacked, has been recently maintained by Professor Hirsch, the celebrated authority on infectious disorders, who, in a discourse delivered last year before the Medical Association of Berlin, expressed his belief that the poison of the plague which broke out at the end of 1878 in Vetlianka in Astrakhan, resided in the packed goods that were being conveyed from Mesopotamia to Armenia; and that, when these goods, having become the spoil of the Cossacks, were taken by the latter to their homes, and there opened out, this poison became diffused.

Pali Plague and Mahamurree.—The questions not unnaturally arise—Are Pali plague and mahamurree identical? And "is it possible that the latter was imported into Kumaon and Gurhwal from the West of India, where the former had been so virulent?" Although neither Dr. Pearson nor myself (nor, I believe, other enquirers into the origin of mahamurree) believe in this importation, I am bound to admit the possibility; and I do so the more readily, because I would gladly defer to the opinion of so distinguished a writer as Dr. John Murray, who states, in the paper above referred to, "this contagious fever spread northward, by Jeypore, to the Doab, extended to Rohilcund,

at the base of the Himalayahs, where Kumaon and Gurhwal are situated. . . . . A pilgrim from Kuttywar, proceeding to Kedarnuth, may have conveyed the infection in his clothes or person, and thus communicated the disease to the head priest and his assistant Brahmins."

Communication between Kumaon and Gurhwal and the West of India.—On referring to the map of India, we find that a distance of between 700 and 800 miles separates the birth-place of the plague in the West of India from Kumaon and Gurhwal, the plains of Northern India intervening: and I am not aware of any regular direct communication between the two. The Bhotiyahs—the carriers of the hills before referred to—annually descend into the plains during the cold season, and visit some of the principal towns there, as Allahabad, Agra, Delhi, etc., going, now that they have, in a great measure, overcome their dread of small-pox, as far as Calcutta. But I believe that they have never yet penetrated into the presidencies of Madras and Bombay. Railway facilities may, in the future, enable them to do so; but, without these facilities, it would be next to impossible, their time being so limited. The Bhotiyahs are always anxious to be back in the hills before the hot weather sets in. But, even if they did traffic with the West of India, the merchandize they bring is conveyed past and through the hill villages, in which mahamurree has appeared, into Thibet, where the bales are opened, and where the plague has never been. I once met with a curious instance of the occasional intercourse that is brought about through the medium of merchandise between remote districts. Goître is not uncommon amongst the Bhotiyahs, who purchase, at a high price, in view to removing it, a semi-leathery substance, which, obtained originally in Surat, is now and then met with amongst the heterogeneous wares of a venturesome itinerant pedlar, who occasionally finds his way into their villages. The Bhotiyahs look upon this as the best remedy for goître in existence. It is taken internally. Curious to know what this thing could be, I put a small piece into a saucer half full of water. It swelled out into a *fucus*, containing, of course, iodine. But even this sort of communication, which would be exceptional, does not account for the systematic importation of plague-poison into those villages of Kumaon and Gurhwal where mahamurree breaks out. If imported at all, the importers would be pilgrims. But pilgrimages are made annually, as heretofore, to the same shrines, the insanitary surroundings being, so far as I am

aware, the same as ever; and yet there has been no outbreak of mahamurree there for more than half a century. Dr. Murray writes, speaking of the outbreak at Kedarnath: "The disease, thence arisen, spread to the villages or religious assignments connected with the temple, and afterwards extended to the villages in the route of pilgrimage." This passage conveys the idea that pilgrims introduced the disease into Kumaon and Gurhwal—an idea, however, which has not been entertained by the local authorities, at any rate, in recent times. I have already remarked upon the personal immunity of the pilgrims in the present day: and my attention was especially drawn to the fact by the local authorities, when I was first associated with Dr. Pearson. That Mahamurree broke out at Kedarnath amongst a body of travel-worn and attenuated pilgrims, in consequence of local conditions then and there existing, one can readily believe; but that a pilgrim conveyed the poison on his person a distance of 800 miles in, say, forty days—giving twenty miles a day as his rate of travelling (sickness or other detaining causes might have made the time longer)—without communicating it *en route* is, I think, with great respect for Dr. Murray's views, less conceivable than that the disease originated from local causes, which are always more or less in operation in these hills—causes which are known to generate virulent disease without any aid from without:—witness the typhus-stricken survivors of the Black Hole tragedy in Calcutta; the outbreaks of the same disease in some jails; and of blood-poisoning in overcrowded hospitals and emigrant ships.

Exportation of Mahamurree.—Whilst discussing the question of importation, we must not lose sight of one that is quite as important, viz., the exportation of mahamurree. Dr. Pearson informs me that he has never heard of it since the time when we worked together. A disease, having very much the same general character as mahamurree, appeared in one or two villages at the immediate foot of the hills in which mahamurree was raging; and the civil surgeon* in charge believed in its having been brought from those hills, and in its being conveyed, through contagion, from one village to another. The infected villages were in an insanitary state, and rats died, though not, as in the hills and at Yunnan in Western China, in the first instance. They died after the appearance of the disease amongst the human population.

Whilst, from the past history and general characteristics

* Dr. Stiven.

of mahamurree, we may reasonably infer that there is but little fear of its appearance in localities unsuitable for its reception, the fact of its having broken out in localities that are suitable (aye, even in those that apparently are not —witness some of the cleanliest villages in Kumaon), the inevitable conclusion is forced upon us, that the only safeguard is thorough sanitation in all its forms.

Quiescence of Pali Plague.—The present inactivity of the Pali plague is a point of some interest. For forty-three years it has not been heard of. What are we to infer from this? Are the localities in which it broke out before in a more wholesome state than they were in then? Has there been any change in the commercial relations with plague-infected countries? Or is everything in the *status quo ante?* I have solicited the favour of information on these points from the Sanitary Commissioner with the Government of India: but, pending the receipt of a reply, I venture to think that, as no fermentation can take place in saccharine solutions without the fulfilment of the conditions necessary to the setting up of that process, so, doubtless, there is some deficiency beyond our ken in those that lead to the development of diseases like the plague.

Remedial Measures.—The remedial measures within our reach are, in two words, *improved hygiene;* which consists of what is personal, and of what affects the surroundings.

Personal.—As reformation is not very probable in the case of adults in vice, we can hardly hope for any great results by preaching cleanliness to the present generation of Kumaonees and Gurhwalees. But we may educate the young; and suitable books have accordingly been published, for use in the Government schools. The natives of India, generally, are fond of tales that abound in proverbs; and a work of this kind, in two parts, has been specially prepared for these mountaineers. It is called "The story of Furnkhabad and Budrenath". The first part tells all about small-pox and vaccination; the second, of the advantages of cleanliness in warding off such diseases as mahamurree. So well did the authorities think of this little work, and so popular did it become, that, when all the copies were destroyed in the Mutiny of 1857, the Government had it reprinted. The recent introduction of a *Sanitary Primer*, as a text book, into the Government schools, is an important step in the right direction.

Surroundings.—But, whilst we can only recommend personal cleanliness, we can enforce cleanly surroundings. We can, *e.g.*, insist upon periodical whitewashing with lime or

clay ; upon better ventilation ; upon the proper disposal of all kinds of filth and the prevention of decay in it ; upon the curtailment of the low growing vegetation ; upon the cremation of the dead ; and on the burning of sulphur * in anticipated outbreaks of the disease ; and upon the location of the cattle away from human habitations. There is more opposition to this last measure than to any ; involving, as it undoubtedly does, danger from tigers in the wilder parts of the country. Stone enclosures, which are safest, are, if made to be of any use, expensive. Still they (or in districts where stone is less abundant some substitute) should be insisted upon, Government making the expenditure lighter by remitting a portion of the revenue. I very much fear that, until the people themselves become hygienically wise, there never will be any heart in their co-operation with the authorities, and that this Augean stable will remain but half cleansed. When sanitary reform was first introduced, so little did the villagers believe in it that, preferring, moreover, the old groove, they simply disobeyed the instructions of the Government directly the medical officer's back was turned ; and it was only when Dr. Pearson was invested with magisterial authority and power to enforce his own orders that they were complied with. So far, mahamurree has only been scotched ; but the efficacy of sanitary measures has been clearly demonstrated (two or three years after their introduction Dr. Pearson wrote to me saying, "mahamurree is practically extinct"); and we have, therefore, every reason to hope that, as leprosy—once so rife in England—and other pestilential diseases have, with the advancing tide of civilisation, disappeared from our midst, so, when they come to acquire clearer views on the subject, mahamurree may become to the Kumaonee and Gurhwalee as much matters of history as these diseases have become to us.

Medicinal.—From the exhibition of drugs, there has been no result sufficiently satisfactory to justify confidence in any. Hyposulphites, as suggested by Dr. Watson, would doubtless be found useful. Regulation of the secretions, promotion of suppuration in the glandular swellings, aperi-

* When the inquiry was instituted in 1852-53, it was considered necessary to destroy the infected villages by fire, compensation being awarded. But, if the same ground is to be re-occupied, burning sulphur would answer the same purpose. I believe that nothing short of rebuilding on improved principles on a fresh site would be of much permanent use. A combination of *gutting*, destroying the wood-work by fire, and to which the natives themselves are partial, and sulphur-burning (this last, to be effective, must be done *very thoroughly*), would doubtless make the purification of the infected spot as complete as possible.

ents when called for, with general support of the system, is the most promising line of treatment. Whatever benefit might accrue from medicines would be much diminished by the delay in their administration. By the time a medical officer could hear of a case, and reach it, nature would have decided the issue one way or the other. To obviate this disadvantage the people have, from time to time, been provided with a few medicines and a memorandum of instructions how to use them. In preparing this memorandum, the opportunity was taken to draw their attention to the value of certain local remedies growing within their reach.

Summary.—Briefly to sum up. 1. Mahamurree is bubonic plague. 2. It is endemic, caused by an animal poison, which is generated by the operation of local conditions. 3. It is quite distinct from—and, as a rule, in no way modified by—the types of fever that are caused by malaria. The usual malarious fevers of India, intermittents and remittents, prevail in these hills from time to time, but they have no connection with mahamurree. 4. Cholera also appears occasionally, but neither is there any relation, vicarious or otherwise, between it and mahamurree. 5. The cattle suffer in some years from foot and mouth disease, but not, it is said, more in mahamurree than in other years. 6. Mahamurree is highly contagious (I use the term synonymously with infectious); and, once in existence, is rapidly propagated from person to person and from place to place, the development of the disease being limited, however, to those persons and those places where the soil—*i.e.*, the conditions favourable thereto—is suitable. 7. Dry heat and cold, especially the former, are unfavourable to the development of mahamurree, which may appear at any temperature and season, though the poison becomes comparatively inert in the dry, hot weather. It ceases, too, during the heavy rains. 8. When once the disease is established, medicines are of little or no avail. To eliminate the poison, to counteract its virulence, and to support the strength, is the most promising line of treatment. 9. It having been proved that sanitary measures are of decided efficacy, our main efforts should be directed to their rigorous enforcement, by which, the people themselves joining heartily in the work, we can alone hope for the eradication of this plague from the paradise of India.

REMARKS ON EPIDEMIC DISEASES—MEN AND ANIMALS—IN THE COLONY OF NATAL.

By Surgeon-General ROSS, C.I.E., F.R.C.S.

[*Read: January 7th*, 1880.]

The Colony of Natal lies on the south-east coast of South Africa, between east longitude 28° 50′, and 31° 30′; and south latitude 27° 20′, and 31° 5′; bounded on the east by the sea for half its length, then north-easterly by the Tugela and Buffalo Rivers, dividing it from Zululand and the Transvaal; on the west by the Drakensburg mountains, a range averaging from 7,000 to 8,000 feet above the sea. On the north, the boundary comes to a point where the high road crosses the Drakensburg into the Transvaal, the sources of the Buffalo being on the other hand. To the south, a river (Umtam Vuna) separates Natal from Pondoland and other counties. The length from south to north would be about 260 miles, and breadth about 140 miles at the broadest point. Three or more high ranges of hills, spurs of the Drakensburg, cross the colony towards the sea, forming uplands, averaging 3,000 to 4,000 feet above the sea, and midlands—valleys—from 1,000 to 2,000 feet; while the coast lands vary from a few hundred to a few feet above sea level.

There were but few Whites in the country before 1834; then there was a large immigration of Dutch from the Cape Colony. The succeeding years tell of wars between Dutch and Natives, and Dutch and British. In 1842, the British compelled the Dutch to come to a proper understanding, and during 1845 the country became a Crown Colony; which, in 1856, was made independent of the Cape Colony by Royal Charter.

The inhabitants, at the close of 1878, are believed to have numbered 22,654 Whites, and 302,858 Natives, 12,000 being natives of India. The area, in square miles, is stated to be 21,150, giving a population of 1.07 White, and 14.7 Black, to the square mile. There are but two towns: Durban, on the sea, the port of Natal, containing between 5,000 and 6,000 White inhabitants, and about the same

number of Blacks; and Pieter Maritzburg, the capital, fifty-four miles inland from Durban, and said to contain nearly 5,000 white, and about 3,000 black residents. There are many villages and settlements marked on the map which will become towns in the future.

I place these facts shortly before you to show, that with such a recent history of colonisation—between twenty and thirty years—and with such a sparse population, no occurrence of epidemics would be anticipated.

The climate of the colony has proved exceedingly healthy for men, women, and children, and the only reason for my addressing these few words to you this evening, is because of what I cannot but think is an anomaly;—that with such a healthy climate for Whites and Blacks, the country should be so unhealthy, and so subject to diseases and epidemics among animals.

When the colony was at peace, the death-rate among the few troops was 7.6 per thousand. Cholera has never been known. Small-pox, which has appeared in the Cape Colony, and even, mildly, in Zululand, only once caused a few cases on the Zulu border of the colony.

During 1878, however, there was a small outbreak of typhoid fever among the residents of Durban, owing to defective conservancy arrangements, and imperfect water-supply, which are being gradually corrected; and a few scattered cases occurred during the first half of 1879.

While speaking of typhoid fever in the Colony, I may state that I believe typhoid fever was the principal, if not the only disease which affected the troops during the recent campaign in Zululand in any serious form.

Army medical officers have reported many admissions occurring in troops encamped on the uplands, in good situations and with fair water-supply, on grassy lands occupied for the first time, and under good sanitary conditions and arrangements. Other medical officers, particularly those with General Crealock's Division on the coast lands, have reported admissions from camps occupying bad local sites, though all care had been taken in their sanitary conditions.

The disease also affected the troops under Colonel Pearson, shut up in Ekowe. The medical department classifies the fever as typho-malarial, being a fever of malarial type, with rose spots, and well marked lesions of Peyer's patches. I saw so few of the cases myself, that I am only competent to state the above recorded facts, without expressing any opinion. But from what I gathered in the colony, and learned during former years in India, I should say that

there must be two forms of typhoid fever—the one which might be called European, arising solely from sewage contaminations; the other, or more Eastern type, prevalent, perhaps, in certain latitudes where you have the same type of fever, but more due to climatic than to filth causes.

And, now, to speak generally about the diseases of animals—horses, oxen, cows, sheep, goats, poultry, and even household dogs are affected from time to time. Horses are affected by "horse sickness" chiefly, and "glanders" in a minor degree. The horse sickness is described as "anthrax fever, resulting from the action of a poisonous agent injested in the food, impairing vitality, and inducing a morbid state of the system, with congestive inflammation of the lungs, and often external tumours": such is the professional description given by the veterinary surgeon to the colony. There are said to be several varieties, but all are identical in the condition of the blood, and in the lungs and intestines being more or less affected. It is believed to be identical with the "Loodianah disease" of India, which some years ago was very prevalent. It occurs during the summer months, generally beginning about November or earlier, according as the setting in of the spring rains, and lasts until the first frosts appear in April. Mules are also occasionally liable to the disease, and sportsmen have reported a similar disease prevalent among herds of deer. Certain districts and parts of the country suffer more than others; why, has not yet been ascertained.

You may have seen a recent letter from Sir Garnet Wolseley's camp, which said no horses are being employed in Seccaconi's country on account of the extreme fatality of the climate. In the campaign in this part of the country, during the spring and first week of October of 1878, 90 per cent. of the horses died, and Dr. Wm. Russell reports of the few horses now being employed (8th Nov.) "the poor horses are now dying off rapidly of this inscrutable malady". The disease is general over South Africa. Throughout the Cape Colony itself, from whence twenty-five years ago we used to draw large supplies of horses, breeding has almost ceased, from the mortality caused by it.

The causes of it are as yet practically unknown. Each farmer or observer in different parts of the colony will have different views on the subject. It is not considered infectious or contagious, but that the same causes produce it in animals subject to its influence. The professional opinion is that some poison is present in the grass of certain lands: probably parasitic fungi, affecting weeds and grasses

in the same way as smut or rust affects forage and other grains. It does not affect horses who are well stabled, fed on dry food, and never allowed to graze; but these are exactly the conditions on which few colonists can afford to keep horses. Only about 5 per cent. of the affected survive. As the disease rarely attacks a second time, a high value is set on such "seasoned" survivors—"salted" as they are called. Glanders occasionally occurs, but in an obscure form. There are laws in force for its detection and prevention.

One other complaint, commonly known as "squitters", also affects horses, I have reason to know to my cost. It might be called expulsive diarrhœa. A horse will expel copious relaxed stools to the distance of several yards, accompanied by great muscular efforts of the whole trunk. This is caused by errors of grazing, young spring grass, wet grass, water given too soon after corn, etc. The animals rapidly emaciate, and unless great care can be taken, remain unfit for work for weeks.

The diseases affecting oxen are lung sickness, red water, and quarter evil. Lung sickness is identical with pleuropneumonia. It is highly infectious and contagious, and through affecting routes of travel is fatal and dangerous, as fresh animals are brought under the influence of an infected locality.

I saw at one regular halting place, five loaded wagons drawn up with thirty dead or dying oxen around them from this disease, the remaining, some fifty, having been driven away to avoid further infection. And these wagons would be left until a better season arrived, when fresh cattle would be brought to take them away. The high road through the colony was studded with remains of oxen in different stages of decay. The disease occurs throughout the year, and is not influenced by season; it is more common in certain districts and localities than in others, but is met with all over the colony.

The liability to this disease among breeding stock is one reason of the large extent of acreage required for a stock farm; 6,000 acres is the average size; this would embrace varieties of uplands and midlands, and would enable farmers to move stock from one portion to another. I could find no good account of symptoms or *post mortem* appearances of this particular disease; but, I believe, both would be identical with the complaint as known in Europe.

These colonial oxen would seem to have but little vitality. Correspondents with the army in Zululand have related the deaths of several hundred transport cattle during one stormy

cold night; and I, myself, saw, in the opening hailstorm of the rainy season, which occurred about the middle of September and lasted only a quarter of an hour, three or four dead or dying oxen left on the ground, in each of two teams of sixteen oxen, which were attached to wagons, and were taking stores to the front.

Cows thrive in their own particular localities only, where to man there seems no appreciable difference in climate or food. Of fifteen cows imported by me from the Cape to Durban, in August and September, which were very fine animals, and far superior in size and breeding to the coast cows of the colony, only two were alive by the end of October. Similar mortality attended the importation of a flock of goats. Professional and the best local advice as to the housing and food were followed in both instances, and no one could tell what caused the deaths.

Redwater, or hæmo-albuminuria, as it is professionally called, is a blood affection, attended with more or less derangement of kidney, liver, and stomach, in fact, of all the digestive organs. The primary seat of the disease is not yet certain. Redwater is most virulent, fatal, and prevalent after the rains of the early summer, about September, when the young grass is springing up, and it is most prevalent in very wet summers. The greatest mortality occurs along the principal highways; but all parts of the colony are liable to be affected, though some more frequently than others. Three causes for it are given; but about each there is considerable uncertainty.

1. When cattle feed, in hot weather, on grasses, etc., containing an excess of water, with small flesh or fat-forming materials. 2. When cattle feed on cold, wet lands, on which the grasses contain parasites or fungi. 3. From bad quality of drinking-water, such as that containing decomposed vegetable or animal matter.

"The causes, whatever they are, continuing in operation, those organs which at first were only functionally deranged become organically diseased, great prostration ensues, and death follows." The duration of disease is from five to six days, depending on the severity of the febrile symptoms. I cannot find any good account of the actual symptoms. It cannot even be positively stated whether the colouring matter in the urine is blood.

The disease is stated to be neither infectious nor contagious, in the ordinary meaning of the terms; but it is known to be highly communicable, and healthy cattle should not graze on the same lands as the diseased. Colonists believe the only prevention is to inoculate the tail

with the virus of the disease—whatever that virus may be, which I could not ascertain. You meet many oxen with only half a tail, the end dropping off after the operation; but Kaffirs are so fond of their cattle, they would rather see them die than tailless.

I may state that "hæmaturia is known to be an endemic disease, prevalent among the inhabitants of the Cape Colony, Natal, and the Mauritius, and due to the parasite, bilharzia hæmatobia."

In 1877, a very rapid and destructive epidemic occurred among cattle in Natal, called "Quarter Evil", but of which there is little evidence attainable. It would appear to be a species of malignant anthrax, affecting legs, shoulders, loins, and other parts of the trunk. The disease was reported not to be contagious; but that the same predisposing causes were in operation on all the animals exposed to its influence.

Among *sheep*, what is called "heart-water" is very prevalent and fatal in certain seasons. I have met with no particular description of it, except, generally, that there is fever, and effusion into the serous cavities, and it is said to be caused by bad pasture, dry seasons, etc. More recently, internal parasites have been detected, which I believe have been sent to England for identification. Scab formerly caused great destruction of flocks; but stringent measures were adopted by law, with very good results.

Of poultry I need only say, that a very fine breed exists in the colony; the Kaffirs breed fowls largely. But among them, also, "a sudden and mysterious disease" frequently breaks out, which is called "fowl sickness", but of which no one I met could give me any particulars. A poultry yard is liable to be emptied by it in forty-eight hours. The Government of the country has given attention to these epidemics.

There is a colonial veterinary-surgeon, who, in one of his recent reports says: "Further experience enables me to state that the diseases affecting the different animals depend less on the peculiarities of soil and climate, than on the want of a good system of management. Many of the diseases depend on circumstances which may be modified by care and attention." He entertains no doubt that with the growth of the colony, consequent improvement in agriculture, and continued attention to the knowledge of these diseases, and the best means for their prevention, that much will yearly be done; and that increased knowledge, and united action, will add immensely to the wealth of the colony, and relieve farmers of much loss and anxiety.

ON SOME STATISTICAL INDICATIONS OF A RELATIONSHIP BETWEEN SCARLATINA, PUERPERAL FEVER, AND CERTAIN OTHER DISEASES.

By G. B. LONGSTAFF, M.A., M.B., Cert.Prev.Med.Oxon.

[*Read: April 7th*, 1880.]

About a year and a half ago the idea occurred to me that by plotting out by the graphic method the annually varying death-rates from various causes in England and Wales, as given by Dr. Farr, in the Registrar-General's Annual Reports, interesting results might be obtained, more especially as the resulting curves would allow of easy comparison. It must be well known to all present that results which can only be arrived at with much labour, and some uncertainty, by the study of long columns of figures, become at once obvious when the same figures are reduced to diagrams. The object of my investigations has been, not so much to ascertain the fluctuations in the fatality of the several diseases, interesting as these may be in themselves, but rather, by the careful comparison of the curves, to see whether any, and if so what, relations subsist between diseases believed to be distinct, with a view to correcting, if needs be, the methods of classification now in use, but chiefly to search for ætiological clues.

I have accordingly traced eighty-nine curves, representing the death-rates per million in England and Wales from as many "alleged causes"; the figures being obtained from a table in the "letter of Dr. Farr to the Register-General" at the end of the annual report. In my smaller diagrams, of which I have a specimen here, the facts are represented for the twenty-nine years 1850–78 inclusive. By a simple application of the law of combinations, it will be found that to compare all these eighty-nine curves, two and two together, would involve 3,916 operations. Of these I have as yet only actually made 1,425, leaving still no fewer than 2,491 to be performed. However, as the zymotic diseases have been compared with nearly all the others, there is

reason to believe that the most important results have been obtained already, and that the remainder of the work will be less fruitful.

I should remark, that several of the causes of death have not been examined at all, since the numbers are so very small, that they could not, by any possibility, yield trustworthy results.

It is my intention this evening to put before you, very shortly, certain of the indications of the above 1,425 comparisons. A variety of circumstances have interfered with my work, more especially the great time required for the construction of these enlarged diagrams; so that I must ask you to consider this as a preliminary paper, which may, or may not, be followed by others, according, on the one hand, as you may be disposed to hear more from me on the subject; and on the other, according as further work may lead to results of importance, or the reverse.

At a very early stage, the close relationships of the curves of diphtheria and croup caught my attention, and soon after the practical identity of the curves of puerperal fever and erysipelas. More recently, I observed that the disease curves may be divided into four groups at least, viz.,—I. The diarrhœal group, characterised by having the greatest mortality during the summer quarter, and by being most fatal in years with hot and dry summers. II. The bronchitic group, characterised by having the greatest mortality during the winter months, and being most fatal in years with very cold winters. III. The scarlatinal group, under consideration to-night. IV. A provisional group, comprising all the other diseases which do not appear, so far as we know at present, to be greatly influenced, or at all events mainly regulated, by any meteorological conditions. Among these may be mentioned small-pox, measles, whooping-cough, phthisis, and (probably) enteric fever.

Remember, that this classification is provisional, and is only drawn up to facilitate the examination of the death-returns of the United Kingdom. Thus, for instance, I know full well that whooping-cough is most fatal in the early months of the year, and that if whooping-cough breaks out during cold weather, it is more likely to be fatal than under the reverse conditions; but what I do assert is, that taking a long term of years, and the whole kingdom into account, the mortality from whooping-cough does not vary directly with the coldness of the winters, as that of bronchitis or pneumonia does.

Since 1855, the Registrar-General has given, in the form of a supplementary table, the deaths in England and Wales in each year, classified under very many more heads than are shown in the other tables. To insure the greatest accuracy attainable, I have abandoned my first charts, and am now working from the "supplementary tables", the labour of calculating the rates being reduced to a minimum by the aid of M. Thomas's Arithmometer—a most useful machine. I soon found it impracticable to draw all the curves to one vertical scale, and finally decided on the method employed by Messrs. Buchan and Mitchell,* viz., of finding the mean for each disease, and plotting out the annual death-rate at so much per cent. in excess or defect of this mean. This entails, of course, additional calculations; but the results obtained are strictly comparable, and enable us to note with ease such facts as that two diseases are affected in the same manner by hot summers, but the one threefold as much as the other: so that it is, perhaps, allowable to infer that, in the former, summer heat is a main cause of the disease; while, in the latter, it is but one of many contributory causes. By employing in all cases death-rates per thousand living in each year (or per million, as is more convenient when the death-rate from a single disease is under consideration), all fallacies due to increase of population are avoided.

The larger diagram accompanying this paper shows certain curves constructed in the manner above described, and grouped together in order to bring out as clearly as may be the points to which I wish to draw your attention. The vertical columns divide the curves into twenty-four portions, corresponding to the twenty-four years, 1855-78. The horizontal portions of the coloured lines mark the height above or below the mean of the death-rate in question in each year—on one scale throughout, each small division corresponding to 10 per cent. The horizontal lines are connected by sloping lines, to give continuity to the curve, and to facilitate comparisons.

The mean of each disease is indicated by a dotted line of corresponding colour; the figure at the right hand showing what is the mean death-rate per million living in England and Wales; while the names of the diseases are given on the left hand in corresponding colours, viz., pyæmia, puerperal fever, erysipelas, "rheumatism, with disease of heart or pericardium", scarlatina, laryngitis, quinsy, croup, diphtheria, and cynanche maligna.

* Journal of the Scottish Meteorological Society, 1874, p. 187.

It will be seen that the curve of scarlatina presents four marked elevations, or periods of epidemic fatality, separated by three equally well marked depressions, or periods of minimum fatality. In the most fatal year (1863), the deaths from scarlatina exceeded the mean by 73 per cent.; whereas, in the least fatal year (1861), they fell to 47 per cent. below the mean.

These four periods of epidemic fatality are, to some greater or less extent, apparent in all the ten curves, the fluctuations varying more in degree than in direction.

In the curves of puerperal fever, erysipelas, pyæmia, and "rheumatism accompanied with heart affection", the fourth or last epidemic period, 1874-5, is the most marked; whereas, in the curves of cynanche maligna, diphtheria, and quinsy, the first epidemic period is far the most prominent.

The second epidemic period is most distinct in scarlatina, croup, and diphtheria; the third in scarlatina and "rheumatism with heart affection", being but faintly indicated in most of the other curves.

Cynanche maligna and quinsy would not be sufficiently important causes of death (only averaging 17 and 14 per 1,000,000 respectively), were it not for their very interesting relations to diphtheria.

Previously to 1855, but few deaths from diphtheria were registered in England and Wales, and they were not kept separate in Dr. Farr's analysis of the Registrar's returns.

In 1856, the total number of deaths registered in England and Wales, as due to diphtheria, increased from 186 to 229—not by any means a striking rise—but in the same year the deaths attributed to quinsy rose from 371 to 413; whereas those assigned to cynanche maligna rose from 199 to 374, a very notable increase. In 1857, the diphtheria deaths rose further, from 229 to 310; those attributed to quinsy from 413 to 485; but those attributed to cynanche maligna increased suddenly from 374 to 1,273, such a leap up as must have alarmed the few persons who were cognisant of the fact. This may be taken as probably the first indication of the coming outburst of what was long spoken of as "the new disease, diphtheria", though, probably, in reality, but an old foe under a new name. In 1858, practitioners began to be more alive to the real facts of the case; and although the deaths attributed to quinsy and cynanche maligna showed still further increments, viz., from 485 to 623 in the one case, and from 1,273 to 1,770 in the other, the deaths more correctly assigned to

diphtheria rose with alarming suddenness from 310 to 4,836. In 1859, the great stir created by the letters in the public and medical Press, drew the attention of all observers to "the new disease", and, accordingly, we see that in that year the curves of quinsy and cynanche maligna began to fall more rapidly even than they rose; and, with the exception of the period of the recrudescence of the diphtheria epidemic in 1863, have continued to fall, till those diseases now have a very insignificant place in the list, and only account for 216 and 179 deaths respectively, on the average of the last five years.

But, meanwhile, diphtheria rose, in 1859, from 4,836 to 9,587. The epidemic may be said to have lasted with diminished, but varying fatality, till 1865. During the last five years, the deaths have averaged 3,092, or about one-sixth of the number that have been slain by scarlatina. It must be borne in mind, however, that the disease is far more prevalent in the United States, in Germany, and more especially just now, in Russia, than it is with us.

The croup curve seems to have been affected in like manner, but in the earlier years to a less extent, by the epidemic of diphtheria. Since 1861, the curves of croup and of diphtheria have been so very similar, as to suggest that at the very least there is great confusion in the diagnosis of the two diseases, and even to be a weighty argument in favour of those pathologists who maintain their identity.

Pyæmia has no place in the Registrar-General's reports prior to 1862, and there can be little doubt but that the steady rise from that year must be attributed to changes of nomenclature; for if not, what is to be said for our boasted improvements in operative surgery and hospital hygiene? In spite of this steady rise, its resemblance to the curves of erysipelas and puerperal fever is strikingly suggestive.

You have, probably, already observed that the curves of erysipelas and puerperal fever are, practically, identical, with the exception that the increased fatality of puerperal fever in 1874-5, was about twice as great as that of erysipelas. I confess that I find it difficult to avoid the conclusion that they are both due to one poison.

Though the puerperal fever curve has great points of resemblance to the scarlatina curve, it is far more like that of erysipelas; it is also extremely like that of "rheumatism with disease of heart or pericardium". So that we may infer some close relationship to rheumatic fever—a point that I must reserve for further investigation, but may merely remark that the erysipelas curve is still more like the rheumatism curve.

I have here a small diagram,* which attempts to represent the mutual relationships of the various diseases in the large diagram—the distances between the circles being supposed proportional to the closeness of relationship.

Dr. Thos. C. Minor, of Cincinnati, U.S.A., writing, in 1874, on "Erysipelas and Child-Bed Fever",† says, in reference to the deaths from these two diseases in the United States in 1870:—

"1. Erysipelas and child-bed fever seemed to prevail together throughout all the States. 2. Any marked increase, in any one locality, of one disease, was apparently accompanied by a corresponding increase of the other. 3. Where histories of past epidemics, of either disease, were obtainable from any of the States, the apparent connection of the two diseases was noticed by physicians at the time of such epidemics, and remarked on. 4. This relationship indicates that there is an intimate connection existing between child-bed fever and erysipelas, and justifies the inference that in any place where erysipelas is found, there will be found child-bed fever."

Again, referring to an epidemic of child-bed fever in Cincinnati, Dr. Minor says:—

"1. The two diseases, child-bed fever and erysipelas, prevailed at the same time, in the same localities. 2. Where an isolated death from child-bed fever was noted outside of the infected districts, a corresponding death from erysipelas was noted in the same locality. This was almost invariably the case. 3. Infants die of erysipelas shortly after or before their parents die of child-bed fever. 4. A few physicians attending child-bed fever cases and erysipelas cases at the same time, as exhibited by the death-register, were the most unfortunate in their practice. 5. Physicians having large obstetric practices, but who were known to be believers in the close connection of child-bed fever and erysipelas, returned few death certificates from either causes."

Again, on examining the alleged connection of typhus fever and scarlatina with child-bed fever, Dr. Minor found that—

"1. Epidemic typhus is not always associated with an outbreak of epidemic child-bed fever, or *vice versâ*. 2. Epidemic scarlet fever is very seldom associated with an outbreak of epidemic child-bed fever, or *vice versâ*. 3. Epidemic erysipelas is invariably associated with an outbreak of child-bed fever, or *vice versâ*."

* The diagram referred to has not been engraved for the Transactions.

† *Practitioner*, August 1875.

I am at present engaged in examining the relations between the death-rates from puerperal fever and erysipelas, in the eleven registration divisions of England and Wales. This small unfinished chart* shows the curves of six of the registration divisions—which shows that in all cases the two curves are very similar. A relationship which holds true in the several parts of the country, as well as in the entire area, can scarcely be fortuitous. Not only do the fluctuations of the curves agree, but it will be noticed that in the district in which one disease is very fatal, the other is also unusually fatal, and *vice versâ*.

I will now examine the subject from a different point of view. The smaller diagram is reproduced with modifications of detail from an admirable paper by Messrs. Buchan and Mitchell, in the journal of the Scottish Meteorological Society, by the kind permission of the authors. These gentlemen analysed the weekly mortality returns for London for the thirty years 1845-74, and constructed curves showing the average number of deaths from a number of causes in each successive week of the year. The curves are on such a scale that in every case the same percentage deviation from the mean is represented by the same distance above or below the dotted line indicating the mean average weekly number of deaths. A little reflection will show that the increase of population does not greatly affect the result, except that great epidemics in recent years must disturb the curve more than epidemics of like relative extent in the earlier years. Diphtheria was not separated from scarlatina in the London returns until 1861, so that the great epidemic 1859-60 is merged in the scarlatina line.

The coloured lines in this diagram correspond to one year—that is one *average* year, which is divided by the vertical black lines into 52 weeks. The months are indicated by figures at the top and bottom. For the sake of distinctness the areas representing the excess of mortality above the mean are coloured in. The coloured figures to the right hand indicate the mean average number of deaths from each cause in every week in London, and the distance of the continuous coloured line above or below the mean indicates the percentage deviation from that number, one small division of the vertical scale corresponding to ten per cent.

It must be premised that "laryngitis" in this diagram comprises laryngismus stridulus and œdema glottidis (the latter of small account), which have been excluded in the

* The diagram referred to has not been engraved for the Transactions.

construction of the larger chart; also, "rheumatism" includes all cases returned under that heading—presumably many of them chronic joint affections, whereas these cases, along with many others, are excluded in the larger diagram. "Rheumatism, with disease of heart or pericardium", may be fairly regarded as comprising few cases other than acute rheumatism, though of course not including by any means all the deaths due to rheumatic fever.

The colours on the two charts otherwise correspond. Now, in certain respects, all the eight curves resemble one another. All the curves are above the mean from the last week in November to the first week in January inclusive, and they are all below the mean from the first week in June to the second week in August inclusive.

There are grounds for believing that if the deaths from laryngismus stridulus could be separated from laryngitis deaths the curve would be somewhat modified, so as to approach more nearly that of croup. As it is, this curve resembles more closely that of bronchitis than my examination of the annually fluctuating death-rates from these two causes would have led me to expect. Messrs. Buchan and Mitchell state that the sudden depression of the curve in January is almost constant from year to year. May not this be caused by the deaths from laryngismus stridulus commencing to rise to their maximum at this point, as the curves of epilepsy and convulsions do?

Omitting for a moment scarlatina from consideration, and dismissing laryngitis as unsatisfactory, we have remaining six curves having a very striking resemblance one to another, which is more especially remarked in the case of erysipelas and puerperal fever. The scarlatina curve, if shifted about five weeks further on, would be not unlike the other curves. It is conceivable that similar causes may tend to the production of scarlatina first, and later on to the other affections—somewhat as a change of type is frequently observed during the course of an epidemic, notably in cholera.

It should be added, that all of these curves have been modified from those published in the memoir referred to by the use of what is known as "Bloxam's method", whereby, instead of taking the average number of deaths in each particular week, the mean of the average of that week, the week preceeding, and the week following is taken. The method is very useful when the number of deaths, or the number of years is too small to give a smooth curve; inequalities produced in accordance with the law of proba-

bilities are removed, and a close approximation is obtained of the curve that would result from the plotting out of a large number of deaths for a longer term of years.

You will naturally ask, "Is there any peculiarity of season or otherwise which determines or has a share in determining the fatality of this group of diseases in certain years?" As the curves as a rule rise in the autumn and remain high throughout the winter, one is naturally led to suspect that a very hot summer, or a very hard winter, is the determining cause. So far as I have been able to investigate the point, it does not appear that the heat of summer or the cold of winter varies either in a direct or inverse relation to the fatality of these diseases. But when the amount of rain is examined from this point of view, it appears that there is some kind of inverse relationship between the two. The same result is obtained whether the amount of the fall or the number of days on which it falls is considered, the connection being somewhat closer in the latter case.

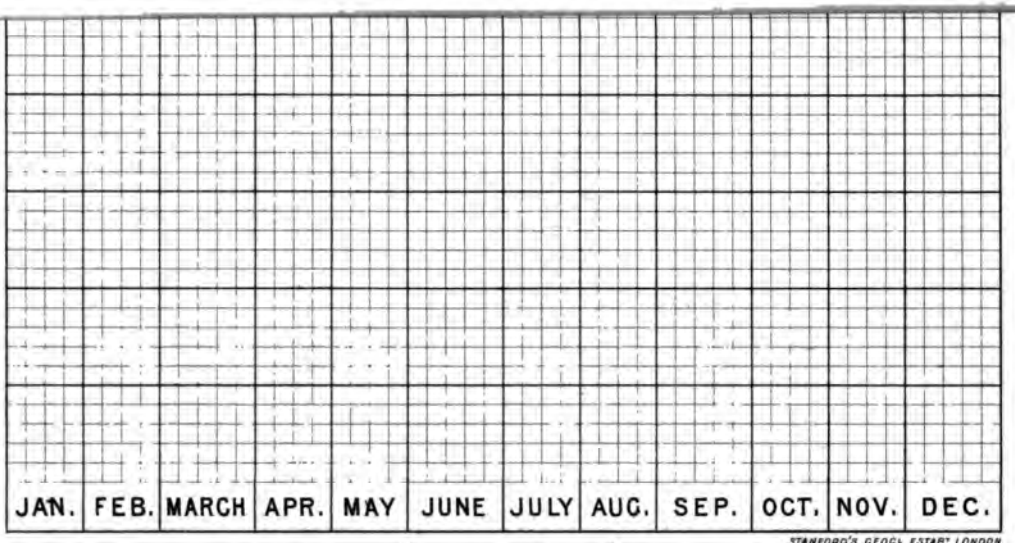
I have calculated the percentage variation from the mean of both the rainfall and the number of rainy days, and added the two together.

The result is shown at the top of the larger diagram, by the blue area, the ordinates being measured from the top downwards, so that the curve formed by the line limiting the blue area is placed upside down, and the inverse relation appears as a direct relation, and is thus much more easily realised. (Dr. Buchanan's method.)

This relationship is nearly as close as that which subsists between diarrhœa and hot summers on the one hand, or bronchitis and cold winters on the other; and it is strikingly suggestive. How the rainfall influences the fatality of these diseases (we do not know how far it may be related to their prevalence) is another question. Assuming the connection to be real, it seems most natural to suppose that the frequent fall of rain purifies the air, and removes from it the particles of contagious matter; that it acts, to some extent, as the process of inunction in scarlatina is supposed to act, by making the poison less diffusible. Anyhow, the seasonal prevalence of these diseases is not at all obviously related to the rainfall.

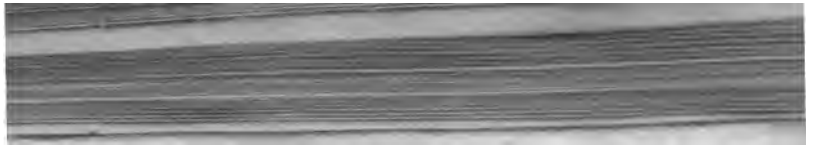
There are many indications, in recent medical writings, of a growing opinion that the process of analysis has been carried too far in our classification of disease; and there can be no doubt that, more especially considered from an ætiological standpoint, such a result is to be deprecated. If great care be not taken, much confusion may be created

as "species making" has caused in zoology and botany. It is important not to lose sight of the possible influence of the individual on the disease that attacks him; further observations on which point are greatly wanted. We may hope for much, too, from increased accuracy in diagnosis, and greater care in filling up death certificates.



The numbers have been adjusted by "Bloxham's Method" previous to the construction of the curves.

Scale. Each small division of the vertical scale corresponds to ten per cent. of excess or defect from the Mean.



EDWARD CATOR SEATON, M.D., F.R.C.P.

By the death of Dr. Seaton the Society has been deprived of one of its most prominent members. As far back as 1840, Dr. Seaton was known as an ardent student of all works relating to Public Health and Epidemiology; and, on the formation of the Epidemiological Society, he at once identified himself warmly with the work it had taken up. Having for many years given much attention to the subject of vaccination, he was appointed, together with Dr. Babington and others, as a member of the Small-pox and Vaccination Committee appointed by the Society, and it is well known that the able report which was issued in 1853, and which formed the basis of the Vaccination Extension Act of that year, was mainly the result of Dr. Seaton's labours. This Report, indeed, ranks amongst the foremost productions of the Society, and its perusal affords ample proof that its principal compiler must have been imbued with a deep sense of that devotion and scientific enthusiasm which, throughout Dr. Seaton's career, characterised his labours in the promotion of that which he knew would constitute one of the highest benefits to mankind. In the year 1857 he published two important papers on the same subject; one entitled, "The Protective and Modifying Power of Vaccination"; the other, "On Public Vaccination in England and Wales".

Indeed, the interest which Dr. Seaton took in securing to the population the more perfect application of Jenner's great discovery, and the time which he devoted to this purpose, were such as materially to prejudice his own professional interests at an important period of his life.

In 1858, he commenced his career as a public officer, and for twelve years he worked, first, as a Medical Inspector, and, later on, as Assistant Medical Officer in the Department of the Privy Council Office and the Local Government Board, at that time presided over by Mr. John Simon. During this period of his life he also issued several important reports on the subject of vaccination. He also

published his well-known *Handbook of Vaccination,* and wrote the article on "Vaccination" in Reynolds' *System of Medicine.*

In 1869, he was unanimously elected to be President of the Society; and, in 1874, he acted as British delegate at the International Sanitary Conference held in Vienna. Dr. Seaton succeeded Mr. Simon, as Medical Officer of the Local Government Board, in 1876. Within about two years of this date there were evident signs that the close mental work he had pursued for a long series of years had seriously taxed his strength, and towards the end of 1879 he was persuaded by his friends to relinquish his official duties. The prospect of well merited rest from work, which had been hoped for, was, however, not fulfilled, for, on the 19th of January 1880, he, at the age of sixty-four, succumbed to an attack of hemiplegia.

In his many able writings Dr. Seaton exhibited that power of clear reasoning which readily carries conviction; during his connection with this Society he not only materially advanced the objects for which it was founded, but also occupied the presidential chair with ability and courtesy; and, in his official capacity, he evinced those qualities which so eminently fitted him for the several posts he filled in so distinguished a manner.

T. RICHARDS, 37, GREAT QUEEN STREET, W.C.

INAUGURAL ADDRESS.

SESSION 1880-81.

BY SURGEON-GENERAL SIR JOSEPH FAYRER, K.C.S.I., M.D., LL.D., F.R.S., Q.H.P.

(*Read: November 3rd*, 1880.)

BEFORE we proceed to the regular business of the first meeting of our Thirty-first Session, it seems right that I should make a few preliminary remarks on the actual condition of the Society, and on the work that it has done during the past year. But let me first thank you for reappointing me to the office of President; and repeat that I shall endeavour to discharge, as efficiently as possible, the duties of the trust you have been pleased again to repose in me.

I am happy to congratulate you on the continued prosperity of the Society, and on the results of its working during the past Session. The increase in number of members, the substantial addition to the funds, and the character of the subjects that have been discussed, are such as may, I think, give cause for satisfaction both as to our present and future prospects. The Epidemiological Society ought to exercise a wide and beneficial influence on the important questions regarding the origin and diffusion of disease that are constantly presenting themselves for consideration; and I believe we have grounds for hoping that this expectation is in some measure realised, and that its influence is gradually growing and becoming more widely diffused; as I hope to show by a brief reference to the subjects that have been discussed, especially those which have given opportunity of comparing certain diseases in tropical climates, with the same diseases as they present themselves here. Let us hope that, as in former years it did in respect of small-pox and vaccination, so it may, in future, exercise its influence in regard to other questions concerning public health, and the investigation of matters that are still the subject of uncertainty in epidemiological science. It appears we have had an accession of twenty-three members during the past year, the great proportion being officers of the Navy, Army, and Indian Medical

Departments; only six were unconnected with the public service.

Whilst fully recognising the advantage arising from the co-operation of those who have had opportunities of studying disease on a large scale, and under varying conditions in different quarters of the globe, I must say, and I think you will agree with me, that we should be glad if our ranks were more largely recruited from the profession generally, and also from other sources. Our Society is essentially catholic in its aims, and its object is the study of epidemiology on the broadest general principles, and under all conditions in which disease appears. It seeks information from medical men or others, whether in the public services or among the large body of physicians and surgeons, as well as other educated persons, who are scattered over the cities, towns, and villages, and rural districts of our own islands, and who are all now more or less occupied—or, if not occupied, are interested—in the prevention of disease and conservation of health.

Therefore, whilst trusting that we may still continue to be supported as much as ever by the medical officers of the public services, I venture to hope that we may have large additions of those who have adopted the private practice of the profession; and I would add that the Society by no means desires to restrict its privileges to medical men, but would gladly associate with it military and naval officers, sanitary engineers, architects, and members of other cognate scientific professions, as well as magistrates or administrative officers, especially those who, in our foreign possessions, have public duties that bring them in contact with questions bearing on public health.

Among the numerous scientific societies in London having claims on medical men, the Epidemiological, I fear, offers comparatively few attractions to the profession generally, concerning itself with questions that are chiefly of general, rather than special, interest. It commends itself naturally to the medical officers of the public services, dealing as it does with subjects that have occupied so large a share of their attention; but it perhaps fails to interest the younger and working class of medical men, who are intent on special subjects of inquiry. But I hold that the Epidemiological Society should include in its rolls *all* classes of the profession, as well as other scientific men, as the questions that fall under its consideration are of interest to everyone; and I hope that, as time advances, it *will* attract such a share of attention as may place it in the position to which I think it is entitled.

The annual report informs us that our financial position, though not one of affluence, is at least fairly good. We have been able, after the payment of all expenses, and after publishing Part ii, vol. iv, of the *Transactions*, at a cost of £31 4s. 6d., to show a balance at credit at the end of the Session of £37 7s. 10d., and it is now intended to commence a new series of *Transactions*. We have also printed three papers on fever in subtropical latitudes, to appear in Part iii of vol. iv. The expense of this and other communications, there is every reason to hope, will be met by the subscriptions of this year. And here we ought, again, to express our thanks to the authorities of University College, for allowing us to hold our meetings in their Council room, free of expense, except such as attends the lighting of the rooms. It has, indeed, been questioned, whether it might not be expedient to revert to the room in Chandos Street, formerly occupied by the Society, and probably the subject may be reopened at some future period; meanwhile the Council have deemed it better to remain where we are; so we shall continue, for a time at all events, to avail ourselves of the hospitality so graciously extended to us, and for which we make grateful acknowledgment.

So far, all I have had to say speaks of prosperity and progress,—increasing numbers, interesting communications, and financial improvement. I have still to refer to the work of the past session, but before doing so, I have another duty to perform. It has been with us as with others—time has brought loss as well as gain; and I have now to remind you of losses too grievous to be repaired, for we miss from among our comparatively limited number, three men of great distinction, removed while yet in the vigour of intellectual life; one at a comparatively early age, just when the promise of his early years was being fulfilled. The past year told heavily on most of our scientific societies, and many an honoured name has disappeared from their rolls, but I doubt if any have suffered, in proportion to their numbers, so much as we have, in the loss of Dr. E. C. Seaton, Mr. H. Leach, and Dr. E. Goodeve. Dr. Edward Cator Seaton was one of the founders and presidents of our Society, and it was during his early connection with it that he commenced the researches into the subject of vaccination and small-pox, which are embodied in his valuable reports, and ultimately led to that important measure, the Compulsory Vaccination Act of 1853. It is in connection with this that his name has attained a wide reputation, for certainly no one has done more towards promoting the diffusion of Jenner's great discovery,

and in systematising the methods by which it has been so largely extended to the people. His writings on vaccination and small-pox are well known as authoritative works on the subject, in regard to which his name will stand on record as that of a public benefactor. Dr. Seaton's public services were not confined to his efforts in respect of vaccination. As an inspector, under the General Board of Health, he was much interested in, and actively concerned himself with, other sanitary subjects; and his selection as the British representative at the Sanitary Conference, held at Vienna in 1874, of which he made a valuable report, bears witness to the esteem in which he was held as a sanitary authority. In 1876 he succeeded Mr. Simon, C.B., as medical officer to the Local Government Board, to which he had previously been assistant medical officer. His extensive knowledge of sanitary science, his administrative capacity and sound judgment, made him a peculiarly fitting successor to his distinguished predecessor. Dr. Seaton enjoyed good health until a year or so after he became medical officer to the Board of Health, when signs of impairment appeared. The duties, which were very arduous, had severely taxed his strength, but he continued to perform them for a year longer, when his medical adviser deemed it expedient to enjoin rest and change; which advice he prepared to follow. About this time a severe domestic affliction made a deep and profound impression on him, and must have aggravated the depression of his general health. He returned to work, however, in June, feeling anxious to resume his duties, in which he might find that solace which perhaps is the best remedy for such a sorrow. Some improvement in his health followed, but it was only transient, for, in the following October, when on a visit in Warwickshire, he was seized with an attack of hemiplegia, from which he partially recovered, and hopes were entertained that his valuable life would be prolonged; but they proved illusory, for he sank under a second attack, on the 21st of January, 1880, in the sixty-fourth year of his age, deeply lamented by his family, and by a large circle of friends. I had only recently made his acquaintance, but was, as others must have been, much attracted by his gentle and courteous manner and bearing; and felt that in him we lost a sterling friend, the public a learned physician and sanitarian, and the Government a valuable public officer,—would that I could have added that the public recognition, to which his services eminently entitled him, had been accorded. His valuable life closed too soon, but not too soon

to have left an example, which those who follow in the walk of life to which he devoted himself, may feel pride in attempting to imitate.

It is also my duty to record the death of another distinguished member of the Society, Mr. Harry Leach, who died at the early age of forty-three, after a brief but brilliant career, which was largely devoted to the advancement of public health interests, in the duties of the important office of health officer for the Port of London, a post of which he was the first incumbent, and which was founded, most probably, as the result of his own efforts during the cholera invasion of 1866, when he was most active, as a medical officer of the *Dreadnought*, in instituting a thorough examination of all ships that came into the river, from suspected ports, and in devising measures for the relief and sequestration of those who were attacked with the disease. He was enthusiastic in the pursuit of professional knowledge, and of all that threw light on the subject he had so much at heart; whilst his acquaintance with disease appears to have been sound and extensive. To his efforts and representations the merchant navy are largely indebted for measures that have tended materially to diminish scurvy; and they received their reward in the success that resulted from them, and in the passing of the Amended Merchant Seaman's Act, especially in regard to the use of anti-scorbutics. In his anxiety to benefit others, he took too little heed of his own health, and the fogs and damps of the river, to which he was much exposed in the performance of his duties, contributed to develope disease in lungs originally delicate. A voyage to Natal produced some improvement, but of brief duration, for he succumbed on the 26th November 1879, to the great regret of all who knew him.

Since writing the above, intelligence has reached us of the death of Deputy Inspector-General E. Goodeve, M.B., Honorary Physician to the Queen, at Stoke Bishop, in the sixty-fourth year of his age. He had for some time been in failing health, from some obscure form of cerebral disease, and the end came rather suddenly on the 27th of last month. He was at one time an active member of our Society, and took a prominent part in the discussions.* His knowledge of disease was profound, and his contributions on cholera, diarrhœa, enteric fever in India, and the so-called "red fever" of Bengal, were most valuable.

His service in India commenced in 1841, and his whole

* His last public service was as British representative at the Cholera Conference at Vienna in 1866.

career, whether in the field during the Sutlej campaigns, in the large civil station of Cawnpore, where he acquired great experience, or during his long connection with the Medical College and Hospital in Calcutta, as Professor of Medicine, and Senior Physician, President of the Faculty of Medicine, and Examiner in Medicine of the Calcutta University, was most distinguished. He rapidly attained the highest honours and position as a physician and a teacher; whilst his retiring, unselfish, straightforward, and noble character endeared him to all who knew him.

The Medical Officers of India have not been among the least of her benefactors; and none, assuredly, worked more to deserve that epithet than Edward Goodeve. His death will be deeply lamented, and his memory fondly cherished by his Service, and by natives and Europeans alike in India.

I now turn to a brief retrospect of the subjects that have occupied us during the last Session. They have all been important; whilst the discussions on them have been most interesting, especially those on Indian fevers, and I trust they may have the effect of drawing attention to the importance of reconsidering the question, especially as respects the etiology of those forms which, while presenting the phenomena of the enteric fevers of Europe, are, by some, considered to be due to general, rather than to specific, causes. I will briefly refer to the papers that have been read during the past year. Three important contributions on plague have been made. The first was by Dr. Payne, one of the two Commissioners appointed by Government in 1879 to investigate the plague which had been prevailing on the banks of the Volga in the province of Astrakhan in 1878-79. This interesting communication threw much light on the geographical distribution, history, progress, and diffusion of the disease, which the Commissioners consider was Levantine plague; and on the measures taken by the Russian Government for its suppression. They consider that there is no evidence to support the theory that it was introduced by Cossacks returning from the war in Asia, or that it was directly transferred from Resht or elsewhere on the Caspian Sea to Vetlanka, but that everything pointed to the conclusion that the disease had already gained a footing in the district (whether introduced from outside or springing up spontaneously in its soil) before the outbreak at Vetlanka. Dr. Payne remarks, that with regard to the possible spontaneous origin, "It appears that the same reasons which will hold to show that plague is endemic, or springs up without being introduced from elsewhere in other parts of the world seem to

apply here. Actual proof of such an origin would be almost, in any case, unattainable; but the difficulties which beset all theories accounting for its introduction into the district, certainly argue strongly in favour of such a view." With regard to the nature of the epidemic; its character, and its morbid anatomy, and the wide spread of the disease, with special reference to personal communication and the influence of local causes and contagion, the Commissioners had no opportunity of forming any opinion from personal observation, as the disease had died out before their arrival. The information they obtained from those examined on the spot is all they had on which to form conclusions on these subjects; and it is to be regretted that on matters about which wide differences have been expressed, the opportunity had not been afforded, to such highly qualified observers, of making personal investigation.

An interesting summary of the German Medical Commissioners' Report on the same subject was read by Mr. Lawrence Hamilton. Dr. A. Hirsch of Berlin, whose account of the plague in Astrakhan differs considerably from that of Drs. Payne and Colvill, describes it as a malignant contagious disease with all the characters of Oriental plague, sometimes with buboes, sometimes without them. He and his German colleagues attribute the outbreak to the importation of infected articles from the Asiatic seat of the Russo-Turkish war, and consider that it was not imported from Resht, neither is it indigenous to Astrakhan, where it had not appeared since 1808. Thus, a further proof is afforded of the difficulty of tracing out the origin of this disease. The report shows that, owing to faulty and imperfect reports of the disease in Vetlanka, the Russian Government could not, or did not, avail itself of preventive or precautionary measures until the plague had attained its maximum intensity, and was already approaching its natural termination. The paper was an interesting summary of the report, and shewed that considerable difference of opinion exists on the subject of the etiology and mode of propagation of the disease.

Another interesting communication was made by Surgeon-General Dr. Francis on the subject of Plague in India. He pointed out the geographical distribution in Kumaon and Gurhwal, to which it is at present confined, and where it is known as Mahamurree, and describes the characters and habits of the people of those districts, which are all conducive to the development of the disease. Known since 1823, but probably existing long before that period, it has been characterised by glandular swellings, hæmorrhages, and rapid

death. It is endemic bubonic plague, caused by an animal poison, which is generated by the operation of local conditions, and is in no way modified by malaria. Intensely contagious among the people themselves, the pilgrims and visitors who visit the shrines in that country, and those who carry merchandise to Tibet, appear not to suffer. Europeans also escape. The Pali plague, a disease having much the same characters, which was first known in the west of India in 1818, seems to be closely allied to the mahamurree by which it has been, as it were, replaced, especially where insanitary conditions prevail, as they are so prone to do among these hill people who claim a Rajpoot descent. It seems as though some common factors were at work leading to the development of epidemic disease, each, *sui generis*, being brought out under the influence of certain local conditions. It is possible that the disease may have been imported through the medium of the cotton workers, who may have received it by importation from Egypt *via* Surat, in goods; but Dr. Francis is of opinion that the local conditions were sufficient to give rise to it, and that no assistance from without was needed. He attaches much importance to sanitary measures, and considers that it has been satisfactorily proved in the case of mahamurree, that sanitary reform, rigidly enforced, was efficacious in preventing it.

These communications, though of considerable interest as contributions to the literature of Plague, do not add very materially to our knowledge on the important question of contagion and mode of diffusion and propagation. Let us hope that at no remote period it will again be brought under consideration.

Dr. Longstaff read a paper on the relationship between scarlatina, puerperal fever, erysipelas, and certain other diseases, in which he deduced, from the comparative death-rate curves and their annual fluctuations, that the different causes of death should be classified into the diarrhœal group relating to the heat of summer; the bronchitic to the cold of winter; and the scarlatinal, and a provisional group comprising all the other causes of death not known to be much influenced by meteorological conditions. All this was represented in elaborate diagrams and curves. One object of the paper was to suggest a probability that the poisons of erysipelas and puerperal fever are identical, while diphtheria and croup also may be due to but one poison; and to discourage the multiplication of species in the classification of disease. The paper was a valuable contribution to statistical medicine —a mode of studying disease which, pursued on an extended

scale, enables the enquirer to arrive by the numerical method at the nature of the laws which regulate and govern the origin and diffusion of disease.

Cholera has not attracted so much of your attention as in former sessions, but it has not been overlooked; and a most practical paper by your late President, who has probably seen more of the disease than any living physician, occupied attention, and discussion, during the greater part of one meeting.

Dr. Murray prepared a most elaborate set of statistical tables, showing the prevalence of cholera in India since the early part of the century, and in his observations on these tables, pointed out the great value of removal of troops or prisoners to other localities on the breaking out of the disease; and that the objections that have been urged against it on the score of expense, and the risks arising from exposure to sun-stroke, fever, etc., etc., are not such as should forbid a change, which experience has shown to be attended with the best results; an argument that could be endorsed by many medical officers. The measures recommended by Dr. Murray, which were much what were officially promulgated in 1870 by the government, comprised, along with change, the strict observance of all sanitary and hygienic details, and enforced the selection of high well-drained ground on the other bank, if possible, should the site infected be near a stream. Dr. Murray's experience, and the constant care with which he has studied cholera in every phase and from every aspect, made the paper one of much practical value, and naturally elicited a considerable amount of discussion on a subject that he has made peculiarly his own.

Surgeon-General J. T. C. Ross, C.I.E., who went to Africa as Chief Commissioner of the Stafford House Committee for the relief of the sick and wounded in the Zulu war, read a paper on Epidemic Diseases in Man and Animals in the Colony of Natal; he pointed out that, although the climate had proved healthy for human beings, it had been unfavourable for cattle; and made some remarks on the prevailing forms of disease. Cholera is unknown hitherto, small-pox is exceedingly rare. Typhoid was the principal disease that affected the troops during the campaign in Zululand, and Mr. Ross says, the disease affecting the troops shut up in Ekowe was called typho-malarial fever with rose-coloured spots and affection of Peyer's glands. The diseases affecting the lower animals were those allied to anthrax, diarrhœa, pleuro-pneumonia, and hæmo-albuminuria. Defective food and water are regarded as instrumental in causing much of the disease. The paper was all the more interesting that it was the result of

very recent personal observation by an officer of large and varied experience.

The subject that has most engaged the attention of the society has been that of certain forms of fever in India and other tropical and sub-tropical climates, and several important communications on this subject have been made by men of large and varied experience. Dr. Chevers on relapsing fever; Dr. Ewart on enteric fever; Dr. Don on the continued fever of sub-tropical climates; and Dr. C. A. Gordon on the continued fever of India—have most clearly placed their views before the Society, and have plainly shown that the subject still needs much investigation; and that, however strictly certain views of the etiology of these diseases may be applicable in Europe, a wider scope must be given to the inquiry, in reference to causation, in other parts of the world lying nearer the equator.

Dr. Chevers traced the history, origin, and the gradual extension of relapsing fever in different parts of India, and its appearance in the form of a distinctive fever in the Indian jails; its relation to other diseases, and its affinities with the so-called famine fever of Europe. The subject had also been reported on by Drs. V. Carter, W. G. Hunter, and Cunningham, and the relation of the spirillum as a cause had been discussed by these officers; the result showing that while some authorities regarded the organism as the cause, others looked on it as merely an epiphenomenon devoid of all casual relation to the disease. Dr. Chever pointed out that though it has not appeared in Lower Bengal of late years, that country was subject to its ravages up to and beyond the end of the last century, and that it has appeared among the poor and half-starved inhabitants of certain districts and in the jails. It has been considered by some that the outbreak of relapsing fever that appeared in Bombay in 1877, was not really referable to the famine that affected large areas of Southern India. The etiology of this form of fever, its relation to the effects of famine, and to the spirillum, are still therefore *sub judice*. It is to be hoped that further research will produce reports that may give more light on this subject. The chief interest of the proceedings of the year may be said to have centred in the discussions arising out of the communications by Drs. Ewart, Don, and C. Gordon in respect of enteric fever. The subject has attracted a good deal of attention lately, in reference especially to the health of European troops serving in India and tropical climates. It is only recently, indeed, that the disease has been recognised as a cause of mortality in India, and now it appears from official reports, that the

death-rate from this disease among our European soldiers, especially the young ones, is very high. The question, therefore, that has been so earnestly discussed is this:—are all the cases returned as enteric fever, in which the symptoms and phenomena in life, and the *post-mortem* appearances are similar to those of enteric fever in this country, due to the same causes? that is, are they the result of the contagion of a specific poison generated in the intestine (or its contents) of one person and conveyed to another through the intervention of air, water, or other mediums? or are they not, in some cases, perhaps a large proportion, due to causes of a different nature, referable to what are known as climatic influences, by which is meant heat, moisture, malaria, miasmata; or to water in which certain matters are dissolved or suspended, with perchance the addition of other causes—aërial-telluric—of which we know nothing positively, but of whose existence as dynamic agencies we probably have indication in this and in some other epidemic diseases? As to the existence in India and other tropical countries of a form of fever characterised by certain phenomena and pathological changes, very closely, if not exactly, resembling those that characterise the enteric fever of Stewart, Budd, Jenner, and Murchison, there is a pretty general consensus of opinion; and there are indeed few who have practised long in India or the tropical or sub-tropical regions, who have not treated cases that in all their symptoms and conditions were like the cases of typhoid they had seen at home. But on the question of causation there can be no doubt, as is evident from the papers referred to, that a considerable divergence of opinion exists, and so far as I can make out from communications that I have seen or received from different parts of the world, there is a growing belief that the cause of some of the cases of the so-called enteric fever is to be sought in high temperature, malaria, miasmata, water contaminated by organic matter (not only fæcal) rather than in a *specific poison* generated in the intestine or in the alvine discharges after extrusion from the bowels—and perhaps in some state of body, especially in the young, which is induced by exposure to one or all of these influences, whereby the poison or cause of the fever is elaborated *in the body itself*. It cannot, however, I think, be disputed that the poison of the enteric contagion theory, advocated by the high authority already referred to, must be equally effective in producing fever wherever it occurs, and certainly not less so in India or the tropics than elsewhere. Few, I think, question its potency now; though, if I may be pardoned for saying so, I think it is *possible* that, twenty years hence, even

here this view may be somewhat modified. But, as I have said, some, and they are not inexperienced, are impressed with the belief that the specific contagion theory will not explain the facts in all cases abroad, nor do they see reason for thinking that a certain train of symptoms and phenomena during life, and lesions observed after death, must necessarily be *invariably* due to *one* single cause; but rather that the bowel ulceration, diarrhœa, spots, and a certain range of febrile disturbance in the temperature and so on, may be brought about also by other conditions, such as those already referred to, and which make up what are known as climatic influences. It is, after all, a matter of speculation, and to a certain extent, of evidence, for even in the most indisputable outbreaks of typhoid fever, wherever it may occur, the specific poison has never yet been seen or separated,—not that this is necessarily a reason for denying either its potency or the probability of its existence on the one hand; but that, on the other, it seems equally reasonable to admit the probable efficiency of other causes.

For my own part, whilst freely admitting that enteric fever in India and the tropics may originate as it does here, I cannot resist the impression that other causes may be at work, and that cases of fever which in India are not only called enteric, but present the phenomena in life, and the changes after death, that are characteristic of that disease in England, do occur, where it is more probable that climatic and other local conditions are the cause than fæcal contagion. But I have no desire to dogmatise, and I await further investigations and reports, before I assert that to be a fact, which though at present only an impression, is so strong as almost to amount to a conviction. It is a subject of considerable importance, and may well stimulate further inquiry, as there is reason to believe that it will, indeed has already begun to, receive, and nowhere with more interest than in this Society.

I may not detain you to epitomise the three important papers, but would briefly say that in them the subject has been ventilated and discussed by men of large experience, who have had ample opportunity of forming opinions from *personal* observation; and the very fact that such observers differ in their views as to the etiology of the fever proves to my mind that there is good reason to believe that the whole question of the etiology of Indian fevers needs further consideration. Nor is this a matter of mere pathological or nosological interest, important as these aspects of the question may be, for it involves grave questions of sanitation,—it

may be, the expenditure of large sums of money in regard to the methods of dealing with the preservation of health, and the prevention as well as the treatment of the disease of the European soldier, or others in these countries. It is obvious that it is very important to investigate the etiology, and this, I trust, the discussions of our Society will in some measure tend to encourage.

I must apologise for occupying your time so long, but the interest of the subject will, I trust, be sufficient excuse for what is but a brief commentary on a subject of much importance. We will now pass on to the first subject of the Session.

ON THE OUTBREAK OF PLAGUE IN THE PROVINCE OF ASTRAKHAN.

By E. D. DICKSON, M.D., Physician to the British Embassy, and Member of the Ottoman General Board of Health.

(*Read: November 3rd*, 1880.)

Sir,—I beg to forward, herewith, a summary of Dr. Giovanni Cabiadis' report on the late outbreak in the province of Astrakhan, in Russia, addressed to the Constantinople Board of Health, from Tzaritzyn, on the 4th April 1879.

It was my intention to have sent this report, long ago, to the Epidemiological Society, but ill health and want of time prevented my doing so; I trust, therefore, that it is not, even now, too late to lay before the Society an account of an incident, which, at the time it occurred, created so great a sensation throughout Europe.

The dates specified in the report are generally given for the two styles—old and new; but when this is not done it is then the old style that is meant. As the Russian authorities seem to have been impressed with the idea that the plague, which manifested itself at Vetlanka, was brought from Kars, and that it had been conveyed to Kars from Mesopotamia by a regiment of cavalry, sent up there during the war, I took pains to inquire into this matter; and the result of my inquiries shows that there is no foundation for such an hypothesis to account for the Vetlanka outbreak.

The archives of the Constantinople Board of Health show that the *last attack* of plague occurred at Bagdad on the 28th May 1877, and the *last death* from it on the 17th June of the same year (the deaths from plague from 27th May to 2nd June were twenty-four; from the 3rd to 9th June, three). Acting from this information, the quarantine restrictions imposed on the arrivals from Mesopotamia were suppressed by order of the Constantinople Board of Health on the 10th of July 1877.

Dr. Duca, the Sanitary Medical Officer stationed at Hilleh, informed the Constantinople Board of Health on the 9th April that, having just finished his second tour of inspection

in Mesopotamia, he was able to declare that the public health was good at Kut-el-Amara, Shat-el-Hai, Samawa, and Divanieh, but that at Hilleh he was informed by the army surgeons that occasionally sporadic cases of plague still cropped up; yet Dr. Duca was not able to detect any of these cases.

On the 31st of May, Dr. Castaldi telegraphed from Resht to the Constantinople Board of Health that the outbreak of Resht was one of true bubonic plague. Dr. Bartoletti, Inspector-General of the Ottoman Sanitary Service, called the attention of the Board to this fact, and informed it that neither the Persian nor the Russian authorities had taken any measures to protect their territories against an extension of the outbreak.

Two regiments of cavalry were ordered on the 5th of June by the Turkish Government from Mesopotamia and Kurdistan to the seat of the war. One of these regiments was then stationed at Kerkuk, and the other at Hilleh. Both, however, were to proceed to Bagdad, in order to take their equipments for the war. At the same time that the Commander-in-Chief of the Army-Corps wanted to despatch these troops to the seat of the war, the Governor-General of Bagdad wished to send 2,000 camel and mule-loads of grain by way of Arbil and Diarbekir to Erzerum. Both these demands were peremptorily refused by the Board of Health, but permission was granted to forward the equipments for the Kerkuk regiment, and the camels and mules and their drivers to Salahié, accompanied by Dr. Cabiadis, in order there to perform a quarantine of fifteen days, during the whole of which term all their effects were to be thoroughly exposed to the sun and air, day and night. No troops whatever were to leave Bagdad for the war.

On the 15th of May, Dr. Lanzoni, the Sanitary Medical Officer stationed at Kars, announced to the Board of Health that the health of the inhabitants was good, but that cases of scurvy still presented themselves amongst the garrison troops.

On the 13th of June, Dr. Cabiadis went to Salahie, and Dr. Duca took his place at Musseyeb.

On the 17th a caravan from Bagdad arrived at the Damascus quarantine station, and Dr. Biagini, the Sanitary Medical Officer there, telegraphed to the Board of Health that all the members of this caravan, as well as all the passengers that had come from Mesopotamia, had arrived there in excellent health.

On the 26th of June, Dr. Bartoletti informed the Constantinople Board of Health that, after the 17th of June, all

deaths from plague had ceased at Bagdad, but that ague of a pernicious type had declared itself there, since the overflowings of the river had inundated the land.

On the 10th of July 1877, Dr. Bartoletti informed the Constantinople Board of Health that the public health in Mesopotamia continued to be good, and that four weeks had now elapsed since the last attack of plague there; and he therefore proposed the suppression of the quarantine restrictions imposed on arrivals from that province. The Board agreed to this, and the quarantine was accordingly abolished from that day. The public health throughout Mesopotamia has continued to be good ever since.

On the 30th of June, Dr. Lanzoni informed the Board of Health that the health of the population of Kars was good, and that the scurvy had apparently disappeared from amongst the troops.

On the 15th of September, he informed the Board of Health that dysentery and typhoid prevailed there, and had assumed an epidemic character.

On the 30th, he gave a satisfactory account as to the health of the inhabitants of that town, but added that there were many cases of scurvy and dysentery amongst the garrison troops.

On the 17th of October, Dr. Cabiadis, the Sanitary Medical Officer stationed at Bagdad, called the attention of the Constantinople Board of Health to the cases of glandular engorgements which prevailed in that town. He had seen from one to four of such cases per week, and had observed that they all suppurated, and finally got well. He had seen, moreover, three cases of carbuncle, but all free from fever.

A telegram from Dr. Castaldi, addressed to the Constantinople Board of Health, from Teheran, on 29th of November, announced *daily deaths from plague at Resht.* Several telegrams from Omer Pasha (*alias* de Gerstdorf), a renegade German officer who commanded the cavalry regiment that went from Kerkuk, by way of Salahié, to Erzerum, addressed to his father-in-law, Dr. Mordtmann, at Constantinople, dated from Bagdad on the 4th and 15th of July, and from Erzerum on the 21st of August, make no mention whatever of *plague*, but only of ophthalmia.

I have the honour to be, Sir,

Your obedient humble Servant,

E. D. DICKSON.

Summary of a Report addressed by DR. GIOVANNI CABIADIS *to the* CONSTANTINOPLE BOARD OF HEALTH, *on the Outbreak which occurred in the Province of Astrakhan in Russia, between the middle of October* 1878, *and the end of January* 1879.

Dr. Cabiadis begins his report with a topographical description of the localities where the epidemic occurred. All the places seen by him along the banks of the Volga, from Tzaritzyn to Astrakhan, were very much alike; and what struck him most (at first), was the width and straightness of the thoroughfares. The dwellings in which the natives live are mere wooden huts, and hardly any of them are supplied with latrines; rich and poor all inhabit buildings of the same size and style, and the largest hut does not contain more than three rooms, and the loftiest room does not exceed 268 centimètres in height. Adjoining the hut, there is a spacious yard to hold cows, sheep, goats, pigs, dogs, horses, and poultry, and which serves at the same time as a latrine. Three or four of these huts are usually built round the same courtyard, but each hut is inhabited by a separate family. The huts occupied by the needy classes are very small and low-roofed. Little holes pierced through their sides serve for windows, and admit a faint light, but they are hermetically closed up in winter in order to keep out the cold. The people live in a filthy state, and the air they breathe in their close habitations is stifling and sickening. The soil is alluvial and damp, and the climate marked by extreme ranges of temperature. In winter the thermometer falls to at least 15 deg. below Reaumur's zero, whilst in summer it rises to 37 deg. above it. The natives are of middle stature, and not particularly robust; their skin is of a tawny hue, and their mental faculties are devoid of intelligence. They occupy themselves partly with agriculture and the rearing of cattle, but chiefly with fishing. Attached to nearly every village will be found the "wattaga" or shed containing pits for curing and storing away salt-fish. The stench that arises from these pits is such, that, excepting to those accustomed to their noxious odour, it is beyond the power of human endurance to remain within the sheds for more than a few minutes.

The above details give an idea of the dirty and insalutary conditions in which the inhabitants of those regions exist. The indigent classes subsist on vegetable food, consisting principally of a soup called "Borsei", composed of rice cooked with salted cabbage, and to which fish is sometimes added.

The wealthier classes occasionally feed on meat and articles prepared from milk, (such as butter, cheese, curds, etc.,) but in general they prefer sending these articles to other places for sale, and limiting their own diet to fresh or salted fish.

Account of the outbreak.—When Dr. Cabiadis visited the localities smitten by the outbreak, the malady had already ceased; he had no opportunity, therefore, of examining patients affected with it, and was obliged to gather his information from statements given him by the inhabitants, by the Russian authorities, and by the medical practitioners. Dr. Cabiadis left Constantinople on the 5th of February, and arrived at Tzaritzyn on the 14th. There he found General Count Loris Melikoff, to whom he gave the letter of introduction he had brought with him from the Russian Ambassador at Constantinople. After reading it, Count Loris Melikoff informed him that the outbreak had already ceased, and that for the last nineteen days no fresh case of it had appeared anywhere. He then inquired what Dr. Cabiadis' opinion might be with regard to the malady which had prevailed at Kars during the war. Cabiadis replied that, as he was at that time at Bagdad, he could not know what was occurring at Kars. Loris Melikoff then rejoined that he was not alluding to the maladies which might have existed amongst the inhabitants and Turkish garrison of Kars, but to the epidemic which prevailed in the Russian army. To this remark Cabiadis replied that, in fact, he had heard of an outbreak of typhus having prevailed amongst the Russian soldiers, and he had no doubt but that his Excellency had received an account of it from the Russian Medical Officers. Loris Melikoff added that, in fact, the Russian doctors had given a description of this malady, and had characterised it as typhus, but that he was convinced it was the plague. He now proposed to Cabiadis that he should take a few days' rest before proceeding to the smitten districts, and sent him with an orderly to Professor Eichwald, the Russian Medical Delegate and Superintendent of the Medical Staff. After several fruitless attempts made to obtain an interview with this officer, both on that and on the following day, Cabiadis finally succeeded in finding him in the afternoon of the third day, and met at the same time Professor Hirsch of Berlin, the German Delegate. These two gentlemen put many questions to him concerning the plague that had appeared in Mesopotamia, and more especially made inquiries as to the type the outbreak had assumed, and as to the precautions taken by Dr. Cabiadis in order to protect himself against its infection. Eichwald then told Cabiadis that, as all the delegates were anxious to pro-

ceed as soon as possible to the seat of the late outbreak, and as there was not a sufficient number of horses for them all, he would have to wait a few days; but, Cabiadis having expressed his earnest desire to start as soon as possible, Eichwald consented to take him along with him and Hirsch next day, February 16th; lack of horses, however, compelled Eichwald and Hirsch, at last, to proceed without him, and Cabiadis was therefore obliged to wait until the 17th, and left Tzaritzyn in company with Dr. Scinighiroff and Dr. Sophia Bastoujewa, Eichwald's assistants, and arrived on the same day at Sarepta (a German colony, 30 kilomètres distant from Tzaritzyn) where they spent the night. Next morning, after a ride of 2 kilomètres, they reached one of the lazaret stations of the sanitary cordon enclosing the whole province of Astrakhan. This lazaret consisted of three small huts intended for the performance of quarantine by those persons that came from places within the cordon, but who had not undergone the quarantine at Vyassowka. Vyassowka is 90 kilomètres distant from this lazaret, and is the quarantine station for the inner cordon which embraced the infected localities, and was intended to protect the districts on the northern line of the outbreak. This station consisted of two huts, each containing two rooms for the reception of persons put into quarantine. Besides these, there was a hut of one room for the disinfection of clothing, and another hut for the medical examination of the persons arriving there.

On the 19th of February, after a journey of 26 kilomètres from Vyassowka, Dr. Cabiadis reached Staritzkoi, a village that lies on the right bank of the Volga, 4 kilomètres distant from the river, and 100 kilomètres from Vetlanka, the principal seat of the outbreak. Staritzkoi contains a population of 2,000 souls, and at the time of the outbreak of Vetlanka nine attacks of plague occurred in this village—all of them fatal. The following is the account given of these attacks to Cabiadis by the "kehya", or prefect, and by the sergeant of the police force. "Ulvana Aksenova Tazkova, a woman sixty years of age, went to Vetlanka on the 9th of December, during the prevalence of the epidemic, in order to read prayers over the dead, previous to their burial. She returned to Staritzkoi on the 15th of December, fell sick, and died within twenty-four hours. The symptoms presented by her malady were intense headache, pain in the hypochondria, and thirst. There were seven persons living in the house at the time Ulvana fell ill and died; but after her death, two of her relatives came to stay with them, in order to wash her corpse previous to its burial. A female friend, who had been suffer-

ing a long time from cancer of the lip, also came to the house to take a farewell view of her. All these people died, one after the other, within ten days, except two children who escaped unaffected. All manifested during their illness the same symptoms as those shown by Ulvana, but one of them also vomited blood. It is uncertain whether glandular swellings presented themselves upon any of these cases. The shortest period within which death supervened, from the commencement of the attack, was twenty-four hours; and the longest, four days. On the occurrence of the two first deaths in this house the inhabitants of the village became alarmed at the circumstance, and removed the rest of the family and the two women who had come to wash Ulvana's body into two houses away from the village; but soon after the family came back to its own house, and more deaths having occurred in it, all the surviving members were carried back to the isolated houses where they had previously been put, and there died, except the two children, who escaped the malady and are still living. The woman, however, who was affected with cancer of the lip, returned to her own home, went to bed, and died within two days." Dr. Cabiadis and Professor Eichwald visited the localities where these houses had stood —for the houses themselves had been burned down in consequence of the occurrence of plague in them. They also visited the cemetery where these people had been buried. Dr. Rutkofski, who was sent to Staritzkoi by the Russian Government, on the occasion of the outbreak, arrived there on the 1st of January, after the first four deaths had already occurred. He stopped there until the 9th of February, and was succeeded by Dr. Krüdner.

According to Dr. Rutkofski's statement, Ulvana Aksenova did not go to Vetlanka for the mere purpose of reading prayers over the dead, but also upon commercial business, and brought back with her two parcels containing stuffs from Turkey, which she intended to sell in her native place. Professor Eichwald, therefore—basing his belief on this statement, attributes the outbreak of plague in the village of Staritzkoi to the stuff said to have been brought from Turkey, setting aside, apparently, the fact that Ulvana was taken ill immediately after her return from Vetlanka, where plague was raging at the time.

On the 20th of February Dr. Cabiadis left Staritzkoi for Tchernyjar, a village of 6,000 inhabitants, 25 kilomètres distant from Staritzkoi. Tchernyjar was not affected by the malady. Next day, the 21st, he went on to Nicolskoi, a village on the right bank of the Volga, containing 1,500 inhabi-

tants, and 25 kilomètres distant from Vetlanka. Two attacks, supposed to have been plague, occurred in this village. The following is the account given of them by a police-serjeant to Cabiadis. "Pavloa, a woman seventy-two years old, who had arrived from Vetlanka on the 22nd of December, fell ill and died within twenty-four hours. After her death, her relatives abandoned the house for thirty days, then fumigated it, and afterwards returned to it. Maskina, a woman thirty-nine years old, without ever having quitted Nicolskoi, fell ill on December 26th, and died on the following day. She had no direct intercourse with Pavloa, but her two children assisted at Pavloa's funeral. After Maskina's death, her husband and the two children were put into quarantine in the lazaret for thirty days." According to the account given to Professor Biesiadecky (the Austro-Hungarian Delegate) Pavloa had started for Vetlanka ten days before her death, but was unable to get to that place in consequence of being stopped by the *cordon-sanitaire;* she therefore returned to Nicolskoi, but was refused admittance under the impression that she had gone to Vetlanka. She was accordingly stripped of her clothes and washed with cold water, while the temperature of the air stood at 12 degrees under Reaumur's zero (5 deg. Fahr.), and then sent to Prischib, 15 kilomètres distant, and put into a solitary hut outside the village. From this place she fled and returned to her own home, suffering from cough and difficult breathing.

Notwithstanding her illness, her daughter, through fear of the police and of the inhabitants, hid her amongst the pigs. On the third day she became much worse, and died on the 23rd of December—that is, on the seventh day of her illness. From this place Cabiadis went on to Prischib, a village of 1,500 inhabitants, 10 kilomètres distant from Nicolskoi, and 15 from the Volga. Here he again met Professor Eichwald and the Russian Commissioners sent there to estimate the value of the houses which had been visited by the plague, in order that they might be destroyed by fire and the owners indemnified for their loss. Dr. Cabiadis here remarks that, before leaving Constantinople, he had read in the public newspapers that whole villages had been burnt down by the Government in order to get rid of the pestilential germs, but on arriving at those very places he found that this had not been done, and that the only houses which had really been destroyed were the three houses at Staritzkoi already mentioned by him, and that these had only been burned down two days previous to his arrival there. In Prischib there

occurred sixteen cases of plague, all of them fatal. Dr. Cabiadis classes these cases into three groups:—

First group.—Arina Effrimoff, a woman twenty years of age, native of Vetlanka, but living with her husband in Prischib, went to see her relatives in Vetlanka, on the occasion of the death of her aunt, named Matrona; she stayed at Vetlanka from the 30th November until the 3rd December. On returning to Prischib she fell ill on the 5th, and died on the 11th of that month. Through fear of catching the disease, no one could be found to bury the body; her husband was therefore obliged to perform this painful duty himself, assisted by his younger brother, named Nicola. This family consisted of nine persons, eight of whom died: four fell ill on the 14th, one of them dying on the 16th; and the other three on the 17th. On the 19th another fell ill, and died on the 21st. On the 20th, two more of the family fell ill; one died on the 23rd, and the other on the 24th. Thus, within thirteen days all the members of this family perished, except an old woman named Russanova who ran away.

Second group.—This group consists of four women, three of them Sisters of Mercy, and the other their servant: all of them died of the plague. The Sisters of Mercy went to Vetlanka, during the epidemic, for the purpose of reading prayers over the dead. They remained there six days, and returned to Prischib on the 8th of December. On the 12th all of them fell ill at the same time: one died on the 15th, two died on the 16th, and the fourth died on the 18th of that month.

Third group.—This consisted of four strong, healthy men, chosen by lot to perform the duty of burying the dead. They lived close to the abode of the afore-named Sisters of Mercy. One of them, named Ivan Petroff, seeing that one of the Sisters (Poliakova) was ill and had nobody to attend on her, went to her on the 15th, fell ill on the 18th, died on the 19th, and was buried by his companions. One of these was taken ill on the 20th, and died on the 23rd; the two others fell ill on the 22nd, and died on the 24th of December.

The plague, therefore, lasted in Prischib from the 5th to the 24th of December, and destroyed sixteen persons. The symptoms manifested by them were tremor, constipation of the bowels, bilious vomiting, coma, heat of the head; in some cases, high fever, cough, and headache, and in one case, vomiting of blood. The bodies of the Sisters of Mercy and their servant turned black. On enquiry being made by Cabiadis whether buboes had been noticed upon any of these cases, he was told that the patients were too ill to call atten-

tion to this indication, had it existed, and no one dared handle their persons, from fear of catching the malady. These details were furnished to Dr. Cabiadis by the Serjeant of Police, in the presence of Mr. Constantine Lebedoff, Councillor of State and President of the Commission appointed to estimate the cost of destroying the houses infected by the plague. They tally exactly with the information obtained by Professor Biesiadecky from the old woman Russanova, in as far as relates to her own family, and with that obtained from the local authorities, and from a Cossack, with reference to the rest. Professor Eichwald, however, holds to the following story related to him by the curate of Prischib, who it is said, found upon the altar of his church an old crumpled dress, made of the Persian stuff called "Cannauss". The curate did not know at the time to whom the dress belonged, but was afterwards told by the mother of Poliakova (one of the Sisters of Mercy) that her daughter had brought it from Vetlanka, and had put it herself upon the altar. On hearing this, he removed the dress with great precaution from the altar, and burnt it. Professor Eichwald imagines, therefore, that this dress, although manufactured in Persia, must have come from Turkey, and must have brought with it the germs of the plague. But, granting that it had really come from Vetlanka, and been brought by one of the Sisters of Mercy, what need is there of attributing their illness to the dress, seeing that they themselves had been in Vetlanka at the very moment when plague was raging in that place?

On the 21st of February Cabiadis left Prischib and arrived at Vetlanka. Vetlanka is a *stanitza* or Cossack settlement, situated upon one of the most elevated spots on the right bank of the Volga. It is therefore less damp than many other places in that district; and yet, notwithstanding, it has been decimated by the epidemic. Previous to the outbreak, its population amounted to 1,700 souls, but, when Cabiadis visited it, there were only 1,300. These were all Cossacks, and administered their own affairs themselves, the Russian Government interfering only when called upon to do so. Fish is the sole produce of the place, and as the river is frozen during six months of the year, the poverty of these people may easily be imagined from the fact, that they have to sustain themselves during twelve months with the gains they obtain during the six months they are able to work. Hence, they frequently find themselves deprived of all means of existence, and the Russian Government is then obliged to make an allowance to each family of from three to five roubles, according to the number of persons composing it.

This pittance affords about eleven centimes *per diem*, at the utmost, to each individual. Fish is not taxed at all, and the village possesses a large "wattaga" or shed for preparing and salting it. The climate, dwellings, and streets of Vetlanka are such as have already been described with regard to the other villages in the district of the Volga; vegetation, however, is here very scanty. Dr. Cabiadis thinks that it must have been from Vetlanka that the plague was transmitted to Prischib and to Staritzkoi, as well as to the villages of Michaelowka and Selitrenoi on the left bank of the Volga.

On arriving at Vetlanka, Dr. Cabiadis met there Professors Eichwald, Hirsch, and Petresco (the Roumanian Delegate) and the Austrian Delegate. On the 22nd, Count Orloff Denisoff, the acting Governor of Vetlanka, called a meeting of all the Delegates at his residence, in order to comply with Count Loris Melikoff's wishes that they should forward him a collective telegram expressing their opinion as to the nature of the outbreak which had manifested itself in that country. There were present at this meeting Count Orloff Denisoff, the acting Governor of Vetlanka; M. Pissaref, the Red Cross Delegate; Professor Eichwald, the Russian Delegate; Professor Hirsch, the German Delegate; Professor Biesiadecky, the Austrian Delegate; Professor Petresco, the Roumanian Delegate, and his assistant, Dr. Protopapesco; Dr. Krassowsy, the Russian Medical Officer, stationed at Vetlanka after the first week in January 1879; and Dr. Cabiadis, the Ottoman Delegate. During the discussion which ensued, Professor Biesiadecky remarked that the result of the enquiries made by him at Vetlanka showed that buboes had also been noticed amongst the manifestations of the epidemic. To this remark Professor Eichwald observed, that what Count Loris Melikoff wanted to know was the name to be given to the outbreak. To this, Dr. Cabiadis replied, that from the information obtained by him while at Staritzkoi and at Prischib, the contagious and fatal character of the outbreak was manifest, and its propensity for attacking and destroying successively within a few days all the members of a family, while sparing at the same time their neighbours and the inhabitants of the place in general, shows it to have been one of true bubonic plague—a malady which he had carefully studied, on two occasions, during two epidemics at Bagdad. His convictions were moreover strengthened by Professor Biesiadecky's declaration that buboes had actually been noticed on the patients attacked by the epidemic. All the other delegates seconded this opinion, and it was therefore officially announced that the outbreak which had shown itself in the Province of Astra-

khan, from the middle of October 1878 to the end of January 1879, was one of bubonic plague. Professor Eichwald then said that their mission ought not to be limited to this simple announcement, but that it ought also to endeavour to trace the source of the outbreak. Historical facts, he added, showed that all the previous outbreaks of plague in Russia had their origin in the two neighbouring States—Turkey and Persia. There could not be a doubt, therefore, but that the late outbreak came also from Bagdad, and was brought by the troops sent up to Kars from that province, at a time when the plague was raging there. The Russian Government had, in consequence, protested against such a step, foreseeing the danger which would menace all Europe if this dreadful scourge were imported into Russia. In support of his argument, he read a long report drawn up by Professor Pelikan, President of the Medical Council of St. Petersburg, and proposed that the delegates should affix their signatures to this paper so that the European Governments, basing their opinion upon this document, might compel Turkey and Persia to adopt efficacious hygienic measures to extinguish the sources of that scourge in their countries. He added, that such efforts ought to be made in the name of humanity, in order to give them greater weight and render them more useful to all. But if, on the other hand, the Commission were to decline sanctioning Pelikan's report, its assembling there for the mere purpose of declaring that the outbreak had been one of plague, would lead to no practical results.

Professor Hirsch praised Pelikan's report, and seconded Professor Eichwald's proposal, saying that he was convinced of the truth of his arguments, and that in his writings he had himself attributed a similar origin to the plague. He even took up the pen and was on the point of putting his name to the paper, when Dr. Cabiadis expressed his astonishment that, without having sifted the matter properly, and without even having given time to the Commission to enquire into the details of the malady of Vetlanka (Biesiadecky excepted, who had been there three days) they wished to attribute its outbreak to an importation from Turkey. He further added, that in the month of May 1878, that is, at a time when plague had ceased entirely in Mesopotamia, he had been ordered by the Constantinople Board of Health to proceed from Musseyeb (a station on the Euphrates, near Bagdad) to Salahié (another station on the Euphrates, on the road into Kurdistan), and there put into quarantine the regiment of cavalry which was to go from Bagdad to Kars to

take part in the hostilities against Russia. He therefore protested against the idea of this Astrakhan plague having been imported from Turkey. Professor Biesiadecky took the same view as Cabiadis, and declared that they should not sign Pelikan's report, seeing that the facts elicited up to that moment did not in any way confirm the supposition, that this outbreak had come from Turkey; that they ought, therefore, to follow up the enquiry, and visit Astrakhan from whence it is said that Mavra Pissareff had gone to Vetlanka, ill with the disease, and had given it to Vetlanka. On the results of such enquiry only, could the Commission give an opinion as to the origin of the outbreak, provided, even then, that the facts acquired by them justified their doing so. Professor Petresco seconded Dr. Cabiadis's and Biesiadecky's views, and Professor Eichwald therefore withdrew Pelikan's paper. This discussion having come to a close, M. Orloff Denisoff drew up the following telegram which was signed by all the members of the Commission, and sent to General Count Loris Melikoff:—"Sur l'invitation du Gouvernement Russe d'exprimer leur avis, les soussignés déclarent que la maladie observée dans le Gouvernement d'Astrakhan, depuis la mi-Octobre 1878 (vieux style) jusqu' à la fin de Janvier 1879, était une épidémie de Peste, revêtant la forme qu'on designe vulgairement du nom de Peste levantine bubonique. Les soussignés croient devoir se reserver d'exprimer leur opinion sur l'importation de la maladie jusqu'à ce que des recherches ultérieures aient fourni des données précises. L'épidémie doit être considerée comme éteinte, vu que depuis le 28 Janvier il n'a été noté aucun cas de maladie, ou de mort. On ne peut, pourtant, donner une garantie sûre que la maladie ne puisse reparaître dans une ou plusieurs des localités qui ont été atteintes et specialement à Vetlanka, qui a été cruellement ravagée. Dans les circonstances actuelles, les soussignés croient pouvoir recommander les mesures suivantes, proposées par le Professeur Eichwald.

"1. Surveillance médicale prolongée des lieux suspects.

"2. Maintien des Cordons sanitaires, qui entourent chacun des lieux envahis jusqu'à l'écoulement du terme légale de six semaines.

"3. Prolongation du Cordon général entourant toutes les localités infectes, avec dix jours de quarantaine jusqu'à l'entrée de la haute température.

"En ces conditions, les soussignés ne trouveraient pas nécessaire le maintien du Cordon entourant tout le Gouvernement d'Astrakhan pour préserver la Russie, et d'autant plus

les états limitrophes, d'une invasion, dans le cas que la maladie venait à reparaître.

" Vetlanka le 22 Fevrier (v. s.) 1879.

" (Signé) " HIRSCH.
" BIESIADECKY.
" CABIADIS.
" PETRESCO.
" EICHWALD."

Professor Eichwald now proposed that, before suppressing the sanitary cordon, the Commission should inspect the population of Vetlanka, in order to determine whether cases of plague still existed amongst them, and to take note of the character of the sequelæ left by the outbreak.

This proposal was at once acceded to by the Commission; and Dr. Cabiadis was requested to attend at the inspection, seeing that he had already been present at two outbreaks of the plague in Mesopotamia. The inspection took place four days after this meeting had occurred; and, as Dr. Cabiadis was not sent for to take part in it, he complained of the neglect to M. Alexandre Ossipovicz Pregenof, the correspondent of the *Golos*, and begged him to mention the fact to the Governor. The result was that soon afterwards Professor Eichwald invited him to attend it: the room, however, in which it was to take place was extremely small, and as Professor Biesiadecky wanted to be present during the whole of the inspection, and the other Commissioners had full confidence in him, Dr. Cabiadis voluntarily withdrew from it and turned his attention to making inquiries amongst the local population.

Professor Eichwald was evidently vexed that the Commissioners had not signed Pelikan's report, for, on the 3-15th of March, he said at Count Orloff Denisoff's, and in the presence of Hirsch, Biesiadecky, Cabiadis, and Zuber (the French Delegate), that it was not in accordance with scientific research to elicit the details of an outbreak from the sayings of Cossacks, rather than gathering them through one's own observations and experience; and that the Commission would have done better had it assisted at the inspection of the people instead of listening to the stories of Cossacks. As Hirsch also expressed himself in similar language to that of Eichwald, Biesiadecky asked him upon what grounds had he based his own opinion when he arrived at Vetlanka, and telegraphed to his Government that the outbreak was one of plague, if not on the sayings of Cossacks?

On Sunday, 25th of February, while Cabiadis was follow-

ing up his inquiries amongst the inhabitants, he was suddenly summoned to Count Orloff Denisoff's residence, and from thence proceeded with the Count, Eichwald, some other Russian doctors, and the correspondent of the *Golos* to a large open court, in the midst of which were strewn nineteen fez (Turkish red caps), 304 handkerchiefs of various sizes and colours, three knapsacks, a Turkish brass inkstand, and a valise belonging to a Turkish military saddle. On their way to this place they were met by an immense crowd of people, and as soon as they got there, Professor Eichwald, addressing himself to Cabiadis, spoke the following words: "Dr. Cabiadis, we have invited you to come here so as to show you these articles, brought by the Cossacks from Kars, and which had belonged to the Turkish soldiers that had come from Bagdad. We trust, therefore, that you will no longer deny that the outbreak of Vetlanka owes its origin to these pestiferous articles."

Dr. Cabiadis replied: "These fez have never belonged to the Turkish army, for neither the caps nor their tassels are in accordance with the Turkish army regulations. They are the head-dress of civilians, and must have been obtained at shops, for they are of different sizes to fit persons of different ages, from that of a child to that of an adult; and, besides, fifteen of them are quite new, and have never been worn at all."

Count Orloff Denisoff then declared that he had fought against the Turks, and had seen them wearing similar caps. But Professor Petresco remarked that he too had served in the last compaign, and, according to his experience, the only article which could have belonged to the Turkish army was the saddle valise. As for the fez, he had seen similar caps worn only by the volunteers. Dr. Cabiadis then explained that, on the day previous to the arrival of the cavalry regiment at Salahié, a Sheik and his son, with a hundred followers, came there from a tribe inhabiting a district five days distant from Bagdad; the plague had never visited this tribe, still the Sheik and his followers were made to perform a quarantine of fifteen days. These people were volunteers, and did not wear the fez (red cap), but the kuffieh (a square handkerchief tied round the head with a cord); nor has Dr. Cabiadis ever seen Arab volunteers wear any fez at all. "Besides", he added, "even supposing that these articles had come from Bagdad, by way of Kars, how could they have been the fomites or carriers of plague when, setting aside the quarantine performed at Salahié, it is well known:

1. "That no plague existed at Kars, either amongst the population, or amongst the Turkish or Russian army.

2. "That the Cossacks who came from Kars had spent two months on their way from that place to Astrakhan, had always been in excellent health, had passed through and stopped in many towns and stations, and had communicated with thousands of persons without ever having given rise to any manifestation of plague. After their arrival, moreover, at Astrakhan, they had dispersed amongst twenty-two villages, thirteen of which were in the province of Astrakhan, and nine in that of Saratoff, and yet neither they themselves nor the population of the numerous villages amongst which they had settled had shown the slightest sign of the plague.

3. "That the result of their inquiries had shown that the first case of plague was noted upon Mavra Pissareff, who, they say, went to Astrakhan to see her son, a Cossack, who, she supposed, had returned from Kars. She did not see him, however, because he had not yet come back. Mavra, on that occasion, slept five successive nights in a boat which had come from Nicolskoi, a village on the right bank of the Volga, with ten other passengers. On the third day she fell ill, and sent for her relative, Ivan Haritonoff, who, after a delay of two days, came to her and, finding her dangerously ill, carried her to the Church to receive the Communion, and then put her into the steamer which conveyed her back to Vetlanka on that very same day. As soon as she arrived there she put herself into bed, and died after three days, viz., on the 17th. From this starting point the malady spread from person to person, and from family to family throughout Vetlanka, and from thence to the other villages. What connection," asked Cabiadis, "could there have been between Mavra Pissareff and the Cossacks, or persons who had returned from Kars?"

To this inquiry Professor Eichwald observed: "I presume, then, that Dr. Cabiadis believes that the plague which broke out in the province of Astrakhan evolved itself spontaneously in Russia?"

Cabiadis replied: "That he had no preconceived notions with regard to the outbreak. What he aimed at was to inquire and search for its cause, and if the result of the inquiry showed that the malady had been imported from some other place, he would then have no hesitation in declaring it; but, if these researches proved fruitless, then, rather than bewilder his mind with probabilities, he would prefer to acknowledge his ignorance, and not put forward, as had been attempted, imaginary hypotheses."

With these words ended the display of the incriminated articles, and the people of Vetlanka who had come to witness

them then dispersed. But, as they were returning to their homes, they were heard to say: "So, you see, after all, that it was these accursed fez that brought the plague into Vetlanka, and not Mavra Pissareff as we at first believed."

The following statements, however, were obtained by Dr. Cabiadis, with regard to these fez, from the Cossacks to whom they belonged.

1. Simeon Ivanoff Serkin, native of Vetlanka, said that fifteen months ago he had quitted Vetlanka along with his regiment, and had gone to Kars, which was then held by the Turks; after fighting against them, with no results, they fell back upon Alexandropol, then went to Kurukdara, then back to Alexandropol, then to Milekamte, then back again to Alexandropol, and finally to Kars, which they found, this time, occupied by the Russians. They now left it for Alexandropol, Elizabetopol, Kuba, Derbent, and returned to Astrakhan in August 1878, and found the city in perfect health. His regiment was composed of five hundred and fifty men, all in excellent health, they had lost about fifty men during the war. He did not know if any malady prevailed at Kars, and has never heard a word about plague. He, and many of his comrades, purchased from the shops at Kars, handkerchiefs and fez, all of them quite new. At Kuba (near Derbent) they also bought some shawls. On Sunday, 25th February, they delivered up these things to the police-officer Gavril Maxinovicz, by order of the Governor Count Orloff Denisoff. He also added that at the time Dr. Cabiadis was examining these things with Professor Eichwald, he was himself present there, and recognised his own fez and shawls amongst those belonging to his comrades. As far as he is aware, none of the Cossacks bought any articles of dress from the Turkish soldiers, or from the Bashi-Bozuks (volunteers): but they got all their things new from the shops. Both he and all his family enjoyed good health during the whole time of the outbreak at Vetlanka.

2. Ivan Gregori Sherbakoff, stated that about seventeen months ago he had started for Kars, following pretty nearly the same course as that already mentioned in statement No. 1. They fought the Turks twice, but without result. His regiment consisted of six hundred men, all of whom enjoyed, throughout, good health. Very few of his comrades died during the campaign. He does not know whether any epidemic existed at Kars or at Astrakhan at the time. He returned to Vetlanka four months ago. He bought from the shops at Kars some fez, with tassels and silk handkerchiefs. All these he delivered up last Sunday, February 25th, by

order of the Governor. He, too, was present at the examination of the things, and recognised those belonging to him. He lost his wife two months ago, during the outbreak. She caught the malady in going to see a friend of hers who was ill at the time. As far as his knowledge goes, none of the Cossacks bought articles of dress from the Turkish soldiers or from the Bashi-Bozuks.

3. Ivan Sotnikoff stated that more than a twelvemonth ago, he had gone to Kars. His itinerary was similar to that mentioned in statements Nos. 1 and 2. His regiment consisted of six hundred men, all of whom enjoyed, throughout, good health. Some of them, however, were killed in the three engagements they had with the Turks. He does not know whether there was any outbreak at Kars or at Astrakhan at the time. He purchased a fez from the shops while at Kars, and delivered it up on Sunday, 25th February, at the same time that the others were given up to Gavril Maxinovicz, by order of the Governor. None of this family fell sick during the epidemic of Vetlanka. As far as his knowledge goes, no one bought any articles from the Turkish volunteers.

Dr. Cabiadis intended to interrogate all the Cossacks of Vetlanka who had gone to Kars; but as soon as it became known that he was making inquiries of them, they were ordered by the authorities not to appear before him. Dr. Cabiadis, therefore, remained ten days at Vetlanka, but failed in his efforts to obtain any further information from its inhabitants. This imposed silence he attributes to the arrival there of Eichwald; for Biesiadecky, who had gone there before Eichwald's arrival had found the inhabitants docile and communicative; but, the very moment Eichwald arrived, everything changed, and it became impossible to get any details from them. This state of things lasted until the day previous to Cabiadis' departure. He then told Eichwald, that, as he could not obtain any replies to the questions he had put to the inhabitants, the only thing for him to do was to return to Constantinople. On that very day, however, to his utter astonishment, Ivan Haritonoff, whom Cabiadis had been seeking for a whole week, and whom Professors Zuber and Petresco and Dr. Rozsahegyi, also, had wished to see, sent word to them that he would meet them in Petresco's house. There they examined him, and obtained from him the following statement:—

"Mavra Pissareff, forty-two years of age, started from Vetlanka for Astrakhan on the 9th October 1878, by that morning's steamboat in order to meet her son, a Cossack, who had returned

from Kars. On the same evening, Ivan himself started for Astrakhan, where he went to stay with a friend. Five days after this, that is on the 14th, he was asked to go and see Mavra as she was ill. He found her lying in bed, in a small vessel that had come from Nicolskoi with ten passengers but without any cargo. As Mavra knew the owner of this vessel, she had taken up her residence on board, and had remained there all the time she stayed at Astrakhan. Feeling ill on the third day of her arrival, she sent for Ivan, but only succeeded in seeing him on the fifth day; Ivan, finding her very ill took her to the church to receive the Communion, and then put her on board the steamer which would take her back to her native place, Vetlanka. Ivan positively declared that Mavra had stayed and slept on board the vessel in which he found her, and had not gone on shore anywhere; but that previous to her illness she had received a visit from a Cossack, who had returned from Kars by way of Kuba, Derbent, and Petrofscoe, and who at this moment was living in his native place, Graczewka, 23 kilomètres from Vetlanka, and who has never been ill."

This same Ivan Haritonoff, however, when examined by Professor Biesiadecky on two occasions previous to Eichwald's arrival, declared to him that Mavra had neither seen anyone, nor been visited by anyone at Astrakhan.

According to Ivan, the symptoms from which Mavra had suffered were: severe pain in the head, and in the hypochondria; she also complained of great pain under the armpit, but he does not know if there was any swelling there, because he did not touch her. A few days later he returned to Vetlanka and was told that Mavra was dead. He found her niece, Praskovia Haritonoff, convalescent, and his own brother, Makar, Praskovia's husband very ill; and he died on 27th October. Praskovia Haritonoff, and Pelagia Sautin, nieces of Mavra, attended Makar during his illness, and were attacked by the malady and gave it to their relatives and to their children; and thus these families all perished, save Makar's sister, Onisia, and two of his own children. Mavra's family lived in a separate house from her own and at first escaped the infection; but afterwards two nurses were lodged in this house and they gave the malady to its inmates, and it destroyed every one of them, including also the nurses. The symptoms of the malady, according to Ivan, were: shiverings, fever, headache, pain under the ribs, and in some cases in the armpits and groins; and amongst the members of one family (Belloff's) there occurred also vomiting of blood. He does not know at what period the epidemic

was at its height, because he did not go out of his house. He was told, however, that at one time, and during four days, there died more than thirty persons a day. He is certain that all those who came into contact with the sick, or with the dead, also perished rapidly. The panic was general, and no one went any more to the lazaret. The Sisters of Mercy, who dwelt in that establishment, all died. Those also, who buried the dead, all died. No one could therefore be found to do this office any longer; and such was the consternation amongst the population, that three men had to be brought from Astrakhan, specially for the purpose of burying the dead. They put up in the priest's house, who died, as well as his wife and mother; his son, however, and the three grave-diggers, lived. If any one fell ill who had no relatives to look after him, he was invariably abandoned to his fate. If, on the other hand, he possessed a family, he was taken care of by them, but they all perished in consequence. There were no doctors in the place, but only medical assistants (*esperti*), and these all died. Towards the end of the epidemic, doctors did come at last, but they also all died.

This is the only detailed statement of the outbreak that Dr. Cabiadis was able to get at Vetlanka, notwithstanding that he remained there ten days. At every village which he visited, he was told that Mavra Pissareff came ill from Astrakhan, and that previous to her arrival, no one in Vetlanka had spoken either of sickness or of death. According to the church registry, a girl named Eudokia Buharova, aged seventeen years, had died on the same day as Mavra; but she had been suffering for two years from an undetermined chronic malady.

The following is Dr. Krassowsky's account of the outbreak:

Dr. Krassowsky arrived at Vetlanka on the evening of the 27th (8th January 1879) December. He only saw fourteen patients, and nine of them he characterised as cases of *typhus*, complicated with *croupy-pneumonia;* and he believed that the epidemic had a similar type. He administered to these patients sulphate of quinine, and fancied that it did good; yet, notwithstanding, all of them died. The hygienic measures he recommended were: first, to fumigate the houses with sulphur-fumes; then, air them for seven days; and, finally, sprinkle them with a solution of carbolic acid (10 per cent. strength). The courtyards, the latrines, and the stables to be washed with a solution composed of sulphate of iron, carbolic acid, and water. The dead to be

buried in graves two metres deep, and filled up with three alternate layers of lime and earth.

The official documents seen by Dr. Cabiadis consisted of:

1. The necrological register of the church of Vetlanka, for the months of September, October, and November 1878, and in which the first cases of the epidemic are noted.

2. The register, kept by the municipality (commune) of Vetlanka, of the attacks and deaths that occurred while the epidemic lasted, that is, from the 17 (29) October 1878, up to 13 (25) January 1879.

3. The notes left by the late Drs. Morosoff and Gregorieff.

4. The report drawn up by Dr. Döpner, Medical Inspector of the Cossacks, who went on two occasions to visit Vetlanka, viz., on the 18th November, and on the 5th December 1878.

5. The official telegrams relating to the epidemic.

1. The necrological register for September, October, and November, kept at the church of Vetlanka, gives five deaths during the month of September; six during October; and nineteen during November. Thirteen of these last deaths are put down to *pernicious fever;* all the rest to attacks of diarrhœa, catarrh, and to old age.

2. The municipality register shows 371 deaths; 357 of which were caused by the epidemic (318 upon natives of the place, and 39 upon strangers). Amongst the strangers who died of the epidemic are noted—a priest, his wife and mother, three doctors, six assistant medical officers, and two Sisters of Mercy. The outbreak lasted thirteen weeks. It reached its acme during the ninth week; and the greatest daily number of deaths, viz., thirty-six, occurred on the 16th December. Only sixty-one attacks got well.

3. The notes kept by Drs. Morosoff and Gregorieff (who both died during the outbreak) record seventy cases attended by them in December. Seventeen of these are put down as *typhoid fever,* of which fifteen died; and forty-three are put down as *croupy-pneumonia,* of which forty-one died. The other ten cases (of which five were fatal) were put down as diarrhœa, 2; pleuritic effusion, 1; ulcer, 1; ague, 2; menorrhagia, 3; unspecified, 1.

4. Dr. Döpner telegraphed from Novi-Vremie on the 21st January 1878, that at the commencement of the outbreak of Vetlanka, and up to the beginning of November, intermittent fever had prevailed amongst the inhabitants; but, afterwards, glandular swellings appeared in the armpits and groins. He therefore went to Vetlanka on the 18th November, and saw there eight patients suffering from mild

intermittent and remittent fevers, with suppurating axillary and inguinal glandular swellings. The patients were cheerful, walked about, had a good appetite, and slept well. They had been ill ten to twenty days.

All such cases, according to the declarations of the *Felcer* (medical assistants), had recovered. Dr. Döpner had seen, in 1877, at Vorpost, near Astrakhan, forty cases of a similar affection, and fifteen cases at Kazaci-Bogor. Later on, he learned that a malady of a very fatal nature was prevailing at Vetlanka; he therefore again went there in December, and saw twenty-three patients suffering from the following symptoms: headache; aches in the limbs; shiverings and flushes of heat; meteorismus; tumefaction of the liver and spleen; pulse, 100-120. These symptoms lasted two or three days. In favourable cases, perspiration came on; in unfavourable, the fever recurred, accompanied with delirium, insomnia and restlessness; the temperature of the body rose to 42° centigrade; the tongue became dry; the discharges from the bowels became black, and the urine high-coloured and scanty. Death generally supervened during the first or second paroxysm (seldom during a third), and was preceded by a convulsive fit, or by great prostration. Out of one hundred patients that were taken ill from 27th November to the 9th December, forty-three died, and only fourteen recovered. On the 9th December, the malady assumed a more severe type: violent palpitations of the heart came on, rendering the pulse imperceptible; vertigo, restlessness, spitting of blood, vomiting of blood, prostration, somnolency, wandering of the mind, suppression of urine, constipation of the bowels, showed themselves. On the 10th December, petechiæ began to appear, and the patients exhaled an odour of honey, owing (says Dr. Krassowsky) to their being drenched with honey. Death followed the stage of prostration, and that of somnolency (coma?). From the 9th to the 14th December, all who fell ill died.

At the first inspection made by Dr. Döpner, he styled the malady a *remittent fever complicated with buboes*, such as described by Griesinger. After his second inspection, however, and after keeping a record of ten cases under his own observation, he declared the malady to be *a severe malignant fever of the recurrent type;* but, from the statement made by the Felcer, this *recurrent type* existed also in the affection which Dr. Döpner had seen on the 18th November, and which he had named *a remittent fever with buboes.* Yet, Dr. Döpner believed that the one was a consequence of the other; and that the symptoms noted by him authorised him

to consider the malady as *a malignant typhus*, or as Hirsch's *Indian plague*, or as a new disease partaking of the characters of *plague* and *typhus*.

5. The official telegrams seen by Dr. Cabiadis were:

A. A telegram of the 6th December from Dr. Döpner, announcing that there were twenty-three persons ill at Vetlanka; and asking that they might be isolated from the rest of the population, and that temporary hospitals might be established in that place.

B. A telegram of the 7th December, from Dr. Kok, announcing that the bubonic fever had ceased, but that since the 23rd November a recurrent fever had begun to prevail at Vetlanka. The malady ran a rapid course, caused great debility, and reached its acme on the third day. In favourable cases, perspiration came on; while, in those terminating in death, the paroxysms became more violent, and were accompanied with delirium, high fever, and convulsions. In some cases, there was a continual vomiting, and in some, hæmorrhage from the bowels. Death generally supervened after a convulsive fit.

This telegram further states that the Cossacks of Vetlanka belonged to the regiment which had returned from the seat of the war; that they had been inspected on their return, as well as all the other soldiers who had come back from Asia, and that they had all been found healthy, and free from maladies.

C. A telegram from the Ispravnik (local civil authority) of Notajefsk, dated on the 12th December, stating that Dr. Döpner had declared the malady of Vetlanka to be highly contagious.

D. A telegram from General Foss to the Government, saying that the recurrent fever had changed into typhus.

E. A telegram from Dr. Zvigmann (Civil Medical Inspector), declaring that *the Plague* existed at Vetlanka, and asking for the establishment of a cordon, and the isolation of the sick; that in Prischib four women had died who had come from Vetlanka, where they had gone to read prayers to the dead, and that the symptoms shown by them were vertigo, unsteadiness of gait, like that of a tipsy person, anxiety and restlessness, fever, thirst, vomiting of blood, rapid loss of strength, apathy; and that these women had apparently arrived from Vetlanka on the 12th; two of them had died on the morning, and two on the evening of the 15th; but that no buboes or spots had been observed upon any of them; that, with the exception of these Sisters of Mercy, and the persons who assisted in burying the dead,

all the patients had belonged to one and the same family; and that on the 19th, two children of this same family, and a nurse, had fallen ill with headache, distress in the precordial region, thirst, and fever.

F. A telegram from the Pristoff (Attorney-General) of the first district, dated on the 20th, announcing that the eldest daughter and a nurse (also of the above-named family) had fallen ill.

G. A telegram from Döpner and Zvigmann, dated from Einotajefsk, on the 15th, stating as follows. In the beginning of November, cases of ague were observed (at Vetlanka), marked by two or three paroxysms of shivering, and succeeded by a hot stage, and by swellings of the inguinal and axillary glands, often ending in suppuration. On the 27th November this outbreak assumed a new form; the glandular swellings did not appear, but the patients became affected with shiverings, pain in the forehead and temples, vomiting, intense heat, aches in the limbs, giddiness, headache, altered features, injected eyes, nausea, anorexia, furred and dry tongue; in some cases precordial distress, discharges of blood, insomnia, delirium, and great weakness. These symptoms reached their acme on the third day, in the few cases which terminated favourably.

These rapid pathological changes, the failure of every treatment tried, and the infectious and contagious character shown by the malady, led Döpner and Pleanoff to call for quarantine restrictions. In their opinion, the outbreak of Vetlanka resembled *plague*.

H. A telegram, dated 19th December, from Drs. Morosoff and Gregorieff, addressed to General Foss, saying: In the course of our inspection of the stanitza (of Vetlanka), we found fourteen patients; two suffering from diarrhœa, two from pleurisy, two from typhoid, and eight from ague. No *plague*, however, exists in the place. After the 15th, the severity of the cold increased, and the epidemic diminished considerably.

I. A report drawn up by Dr. Gregorieff, declaring that neither he nor Dr. Morosoff had seen any instance of *plague*, but only cases of *croupy-pneumonia*. Its symptoms were: headache, shiverings, increased temperature of the body, rising up to 40° centigrade, acute pains, cough, expectoration of viscous, rusty or red sputa, dyspnœa, lividity of the skin, dulness on percussion over the chest, bronchial respiration, bronchophony, rattles in the throat, death, generally within six days of the illness.

J. A telegram from General Foss, dated on the 28th,

stating that a careful inspection of the sick had given no indication of *plague*, but had shown the existence of an epidemic outbreak of *croupy-pneumonia*; and adding, that the great mortality of these cases arose from the detestable hygienic and dietetic conditions of the people, and that this explained also the infectious character of the outbreak.

Quarantine measures were not deemed necessary; but, if doubts were entertained as to the nature of the malady, specialists ought to be sent to the seat of the outbreak. This telegram is signed by Nicolski, Gregorieff, Golubeff, and Colonel Pleuoff.

K. A telegram from Pleuoff, saying, that many persons are ill, and that Krassowsky considers the outbreak as one of croupy-pneumonia.

L. A telegram from Krassowsky, dated on the 29th, calling the malady *typhoid-pneumonia*, and stating that he had noted also a case with a sore in the groin, of long standing.

M. Another telegram from Krassowsky, dated on the 31st, announcing a fresh case of pneumonia; one apparently of typhus; one of ague; and two deaths from typhoid-pneumonia. He declares, moreover, that up to that moment, no case whatever of *plague* had shown itself.

N. A telegram from Krassowsky, of the 1st January, announcing another case of typhus, and three cases of a doubtful character. The Vetlanka malady is declared to be a typhus, frequently complicated with pneumonia. The great mortality is attributed to the bad hygienic and dietetic conditions of the people. More than 300 persons were then ill, and 273 deaths had already occurred.

O. A telegram from Krassowsky, of the 2nd January, declares, that the recent cases of typhus were all derived from infected houses.

P. A telegram from Krassowsky, of the 4th January, announces, that at Staritzkoi a similar typhus prevailed.

Q A telegram from Krassowsky, of the 7th January, announces that Dr. Gregorieff had fallen ill on the 6th; his malady not clearly made out.

R. A telegram from Krassowsky, of the 10th January, stating that Gregorieff had died on the 8th, but that no fresh case had occurred.

S. A telegram from Krassowsky, also of the 10th, stating that Dr. Gregorieff's brother, Petroff, had caught cold, had fallen ill on the 9th, and had died on the same night.

T. A telegram from Krassowsky of the 14th January, announces a great many deaths from cold; and that the Sister of Mercy, Dobbreggina, had died on the previous day, of a violent cold.

U. Telegram dated 11th January, from Dr. Polivanoff* to the Minister of the Interior, declares that the epidemic of Vetlanka was undoubtedly one of *plague*. That from the month of October, up to December, it was associated with buboes; but that after that period, it assumed a pulmonary form.

V. A summary report drawn up by Dr. Zvigmann, stating that, notwithstanding the persistence of the epidemic and the uninterrupted presence of medical men at Vetlanka, no detailed and reliable information on the outbreak had yet been given, as fully shown by the tenor of the above telegrams.

The statements regarding its contagious nature were vague; the number of deaths which had occurred in each family, and at what period, and under what circumstances, was not mentioned; clinical observations were altogether wanting; as also the precise date of the commencement of the outbreak.

W. An announcement from Dr. Kok, stating that only two suspicious cases had occurred at the village of Udeminaja—one upon a lad thirteen years old, who fell ill on the 13th December with headache and bleeding from the nose, and who died on the 18th, and who had been staying at Vetlanka until the 8th. The other case was that of his father, who fell ill on the 2nd January, with fever, shivering, quick pulse (85), increased temperature of the body, loaded, white, and dry tongue, anorexia, thirst, headache, mental faculties unimpaired, breathing free, a small swelling, the size of a pigeon's egg, in the right armpit. He attributed his illness to having caught cold during very severe weather. On the 12th January he was still under observation.

Dr. Cabiadis was desirous of visiting Michaelowka and Selitrenoi, on the opposite bank of the Volga, so as to enable him to complete his inquiries as to the history of the epidemic; but the other delegates did not consider this step necessary, because those places had received the malady from Vetlanka, and much time would be lost in getting back from them, owing to the cordon which still existed round them. Moreover, were fresh cases of the malady to break out there, this would render it impossible to quit the place until forty-two days had elapsed after the last case—as proclaimed by authority. They were all, therefore, of a mind to go on to Astrakhan and there follow up the history of the outbreak. Dr. Cabiadis, therefore, accompanied by Professors Biesia-

* From the tenor of a telegram sent by Colonel Xorstow, it would appear that Dr. Polivanoff had not seen any of the Vetlanka doctors.

decky and Petresco, quitted Vetlanka on the 4th of March for Zamian, where they performed ten days' quarantine before being allowed to proceed to Astrakhan. On the 5th they reached the station of Siero-Glasinka, twenty-two kilomètres distant from Zamian. On the opposite bank of the Volga lay Selitrenoi, at thirty kilomètres from Siero-Glasinka. As these two places are in continual communication with each other, Dr. Cabiadis was able to get from Mr. Korinfski, proprietor of the station of Siero-Glasinka, the following account of the outbreak of Selitrenoi. The correctness of this statement was moreover confirmed by Nicola Iegoriz—a police officer, who had been present in that place at the time of the outbreak.

"In the month of December, and during the height of the epidemic, Plaxin, an inhabitant of Selitrenoi went to Vetlanka, together with a friend of his, for the purpose of selling fish there. Plaxin's companion, alarmed at the panic which prevailed there, ran away and returned to Selitrenoi in good health, and has remained well ever since. Plaxin, however, stayed at Vetlanka a few days, but finding that no one bought his fish, and feeling ill, he went away to the neighbouring village of Sassinskoi, where he thought he could dispose of his fish and remain until he was well again. But, getting worse, he disposed of his fish as best he could, and returned to Selitrenoi, put himself into bed, and after a day or two died there. His wife and his son fell sick immediately after him, and both died also. This is the manner in which the malady was communicated to that place."

It is said that the malignant pustule prevails endemically at Selitrenoi, and Dr. Krassowsky characterised the outbreak of Vetlanka as a *typhus complicated with croupy-pneumonia*, and that of Selitrenoi as an epidemic of *malignant pustule*. Other doctors, however, declared that the outbreak of Selitrenoi was decidedly an offshoot of that of Vetlanka,—viz., a *bubonic plague;* and they all maintained that the pustular disease of Selitrenoi had never, *per se*, caused the death of any person.

With respect to Michaelowka, the malady was introduced there in a direct manner from Vetlanka on the 4 (16) January, by a person named Romanoff who, as well as his wife, died of it. Their son also, fell ill and had a bubo, but got well. Dr. Cabiadis and his companions continued their journey, and arrived at 2 p.m. of the same day at Zamian, where they performed ten days' quarantine, and where they were very hospitably received, thanks to special orders having been sent there to this effect by General Loris Melikoff.

This lazaret establishment began its work on the 4 (16) January; and from that date up to the moment of Dr. Cabiadis' visit, it had received 707 passengers; 386 had already quitted it, and 321 were still in it.

On the 9th March, and whilst the Commissioners were undergoing quarantine at this place, a mild attack of plague was announced to have occurred at Vetlanka. As it was a solitary case, and of a mild character, the Commissioners did not think it necessary that they should all go there, but Dr. Kiemann, Dr. Rozsahegyi, and Dr. Klinimberg, were sent to Vetlanka by Professor Biesiadecky to examine and report on the case. The Commissioners learned subsequently that this patient, a girl ten years of age, had had an attack of fever, which had lasted three days, but that she was now convalescent, and that no untoward event had followed the case. Dr. Cabiadis infers from this that it was, in all probability, an instance of those glandular swellings which commonly follow in the wake of an outbreak of plague, and that the febrile reaction which accompanied it was owing to the delicate constitution and to the youth of the patient. The girl herself accounted for the swelling in the following manner. "The removal of the *cordon-sanitaire* was celebrated by public rejoicings at Vetlanka on the 4th of March, and Count Orloff, the Governor, threw on the occasion sugar-plums to the people. While the girl was scrambling to catch some of these, she received a kick from one of the crowd. At the moment she experienced no great pain; but, three days afterwards, pain and a swelling manifested themselves at the spot where she received the kick."

Dr. Cabiadis, and Professors Biesiadecky and Petresco, left Zamian for Astrakhan on the morning of the 15th March, and arrived there on the same day.

Astrakhan is 120 kilomètres distant from the Caspian Sea. It is built upon four islands formed by the Volga, and is intersected by numerous canals, with banks covered with luxuriant vegetation. It has more than forty churches, with lofty steeples towering high above the houses, and giving a singular and picturesque character to the entire scene. It is the seat of the Government of the province of Astrakhan, and has a population of about 60,000 inhabitants, of almost every race and religion, not even excepting idolaters. The principal races met there are—the Russian, Persian, Armenian, Jewish, Tatar, Cossack, Kirghiz, Karsana, and Kalmuck.

The Kalmücks are idolaters, and are so dirty in their persons and dwellings, that you are compelled to keep at a distance from them, in order to avoid inhaling the stench that

emanates from their bodies. They live on putrid fish and dead animals of every sort—not even excluding dogs. Parental affections are unknown to them; and, when even their own children fall sick, they abandon them, to die of starvation, and transfer their *kibitkas* (dwellings) to other places. The kibitka is a sort of low tent, in which the air becomes vitiated and offensive from the excessive filth of the inmates. The Kalmuck lies upon the bare ground, paying no heed to whether it be dry or damp. The wealthier classes, however, sleep on rugs, or bits of carpet; and Dr. Cabiadis remarks, that it is difficult to conceive how human beings can manage to exist in such deplorable conditions. All the individuals belonging to this race are so alike that it is very difficult to distinguish one from the other. The type is marked by a flat face with prominent cheek-bones (*zygomi*), eyes sunk deep in the sockets, and eyelids half closed.

The streets of Astrakhan are wide and straight, and, with the exception of the public buildings and of a few fine houses in the principal thoroughfares, the style of the houses does not differ in the least from that already described with reference to the dwellings of the people, in general, throughout the province. The situation, and the hygienic conditions of those of Astrakhan, however, are if anything even worse. The water-level in the canals is perpetually changing, in consequence of the ebb and flow of the Caspian Sea, caused by the action of the winds: hence, the land is subject to periodical inundations, and the ground remains permanently damp. The houses are low and ill-ventilated; and deleterious emanations arise from the putrefaction of decomposing animal and vegetable matters. The accumulation of filth in the city was so great, that in order to render it clean when General Loris Melikoff visited it, eight hundred cart-loads of dirt had to be carried away daily, and thrown into the river, for fifteen consecutive days previous to the General's arrival there. Another source of the fœtor which pervades this city, arises from the numerous wattagas in it. To give an idea of the enormous quantity of fish that is caught at Astrakhan, Dr. Cabiadis says, that besides the large amount of caviare consumed by the inhabitants themselves, five million kilogrammes of it are annually exported from that place:—viz., one million and a half of the black sort, and three millions and a half of the red sort of caviare. The staple food of the population is fish, both fresh and salted. The climate of Astrakhan is very changeable; in summer, the weather is hot and moist, and Reaumur's thermometer rises up to 37 deg., and even higher; while, in winter, it sinks to 15 deg., if

not lower. Malarial fevers are the prevailing maladies of that city. On the right bank of the river, opposite to Astrakhan, but forming part of that city, stands the suburb of Vorpost, inhabited by a population of 1,700 Cossacks. This was the place from whence the Cossacks sent to the campaign of Kars were shipped, and where they landed on their return.

On the 17th of March, Dr. Cabiadis and Professor Petresco waited on General Rippen, the Governor of Astrakhan, and informed His Excellency that they had come there for the purpose of pursuing their inquiries as to the origin of the late epidemic. They narrated the story of Mavra Pissareff, to whose arrival from Astrakhan the Vetlanka outbreak was attributed; and they begged His Excellency to inform them whether the plague had really existed in Astrakhan at that period. General Rippen replied that he knew nothing of the story of Mavra Pissareff. With regard to the plague, however, he was aware that in the month of May 1877 cases of a suspicious malady had been observed in Astrakhan, characterised by fever, and accompanied by axillary and inguinal swellings. He had seen these cases himself, and had cured them with sulphate of quinine; but the malady was of no consequence, as it was not contagious, and as no one died from it. It subsequently disappeared altogether, and the public health at Astrakhan has ever since been excellent. Dr. Cabiadis then enquired if any medical or official record had been kept of these cases; and His Excellency replied that such record did exist, and had been sent to Professor Eichwald, who was expected to arrive there in a few days, and would no doubt communicate it to the Commission; but (he added) if they were in search of the origin of the plague of Vetlanka, he could positively assure them that it had been brought from Kars along with the effects of the Cossacks; because it broke out immediately after the arrival of those very same Cossacks. Dr. Cabiadis then enquired whether any outbreak of plague had existed in Kars, and whether any of the Cossacks that arrived from that place had been found suffering from the plague. His Excellency replied that an outbreak of plague had existed amongst the Russian army at Kars, but had been named by the military surgeons *typhus*. It was not typhus, however, but true *plague*. With regard to the Cossacks who had returned from the Kars' campaign, although they were found in excellent health, they must have brought the germs of the malady in their clothing. Dr. Cabiadis objected to this hypothesis of the origin of the Vetlanka plague, as the arguments put forward in support of it were weak, and as no notice was taken of two other factors,

that might account for it, viz., the actual outbreak at Resht in 1877, and perhaps also in 1878; and a spontaneous origin in Russia. General Rippen observed that it would be difficult to understand how it could have been imported from Resht in the face of the strict quarantine restrictions which he himself had organised at that time. As to a spontaneous evolution of plague in Russia, he believed it impossible.

When this interview was over, Dr. Cabiadis set about to collect information from the medical men and from the inhabitants; but all he could get was a mere confirmation of the statement made by Eichwald to Zuber, at Vetlanka, viz., "that in May 1877 an outbreak of fever, with buboes, had prevailed at Astrakhan, and had caused a great sensation there, as well as St. Petersburg. Five reports on this outbreak had been forwarded to St. Petersburg in order to give an idea of its nature. Most of the medical men of St. Petersburg, to whom these reports were referred, pronounced the malady to be an extraordinary outbreak, probably of a malarious nature; but three of them declared that it was the *plague*, and that it required vigilant sanitary measures to restrain it, and this was done."

In a printed report, addressed to the Government in 1878, and drawn up by a medico-hygienic Commission, named by the municipality of Astrakhan, it is declared that if effective measures be not taken for the improvement of the sanitary condition of that city, it would become another cradle (*secunda patria*) for the plague; and that 25,000 roubles would be required to carry out such improvements.

This is all the information Cabiadis could obtain during the twelve days he remained in Astrakhan. It shows, however, that an outbreak had actually prevailed in that city during the months of May and June 1877. Various were the opinions formed by the members of the Commission with regard to the nature of this outbreak.

These opinions are consigned in a report, drawn up in German on the 26th of March, by Dr. Sommerbrodt, Professor Hirsch's assistant. At that sitting were present also, General Rippen, and Drs. Zvigmann and Ghiergi, two medical officers of Astrakhan. In this report it was declared unanimously that the outbreak of Vetlanka had been one of *bubonic plague;* that its character had not differed from that usually shown by the plague, but that bronchial complications had manifested themselves frequently during the outbreak, and that it had spread from Vetlanka to the other localities attacked by it, evidently through the influence of its contagious properties. With regard to its origin, the opinions differed,

both as to that of Vetlanka, and as to that of Astrakhan. With regard to that of Astrakhan, Professor Hirsch declared that it was not plague, and yet he could not tell what the malady really had been. Biesiadecky said that, as he ignored what the malady really was, he could neither call it plague, nor anything else. Zuber, Petresco, and Cabiadis believed it to be a mild form of plague.

Eichwald declared that the malady was merely one of simple glandular swellings, mixed up with a good many syphilitic cases. With regard to the Vetlanka outbreak, Hirsch sets aside the hypothesis of a spontaneous origin and says, that if he could be certain that plague had prevailed at Kars, he would not hesitate to attribute the Vetlanka outbreak to an importation from Kars, seeing that the malady had declared itself immediately after the return of the Cossacks from that place. But, as he is not sure that there was plague at Kars, he is therefore doubtful whether the Vetlanka outbreak came from Kars or from Resht.

Biesiadecky rejects the idea of a spontaneous evolution in Vetlanka; and as to its spontaneous origin in Astrakhan, and to its importation from Kars or Resht, he declares that the result of his inquiries does not allow him to form an opinion.

Zuber sets aside altogether the hypothesis of a spontaneous origin with regard to the Vetlanka outbreak; but, taking into consideration the proximity of Resht, and its frequent communications with Astrakhan, he thinks that its derivation might be attributed to Resht. As he ignores, however, whether plague had existed, or not, at Kars, he is unable to determine from which of those two places it really derived its origin.

Petresco discards the spontaneous origin of either the Vetlanka or the Astrakhan outbreak, and believes the malady to have been imported from Resht.

Eichwald was positive that this plague must have been brought from Kars by means of the Cossacks' clothing.

Cabiadis doubted its spontaneous evolution in Vetlanka. He denied its importation from Kars, because the malady had not existed there; but he admitted the probability of its importation from Resht.

The opinions of the Commissioners were unanimous as to the means by which the outbreak had been circumscribed and extinguished, viz., that this result was due to the establishment of the *cordons-sanitaires*.

The Commission, considering that its mission had now ended, embarked, on their way homeward, on the steamer that left for Tzaritzyn, on the 27th of March.

Dr. Cabiadis concludes his report on the late outbreak in the province of Astrakhan with the following remarks. He declares that not having seen the malady, and having obtained his information from imperfect and contradictory statements, it was out of his power to give a faithful and satisfactory account of the outbreak. His report is, therefore, limited to the few scanty facts that came to his knowledge, and to such remarks as his practical acquaintance with plague suggested.

Symptoms.—A clear description of the signs and symptoms of the malady does not appear to exist. Drs. Morosoff and Gregorieff, who fell victims to the epidemic, left no record of the knowledge they had acquired. Dr. Krassowsky, who went to Vetlanka when the epidemic was on the decline, only saw fourteen cases, and these he characterised as typhus complicated with croupy-pneumonia. The only description of the malady that we possess is, therefore, that given by Dr. Döpner. The symptoms he gives are precisely those of the plague, viz., a severe, contagious, malignant fever, accompanied with axillary and inguinal buboes, petechiæ, hæmoptysis, hæmatemesis, and great prostration, followed by rapid death. As Dr. Döpner's stay at Vetlanka was short, he had no time to follow up the clinical history of the cases observed by him, and to give a statistical account of the progress of the epidemic. The same may be said of Dr. Zvigmann's telegrams. It is uncertain whether buboes were a frequent manifestation or not; but, the character which seems to have prevailed, and to have attracted the attention of those who attended the patients, was the bronchial affection. Out of seventy cases entered upon Drs. Morosoff and Gregorieff's clinical register, forty-one are put down as cases of croupy-pneumonia, of which two only recovered; and out of thirty-nine fatal cases, only two survived up to the sixth day. Dr. Cabiadis here observes that, in the outbreak of plague in Mesopotamia, he never met with any case complicated with croupy-pneumonia.

Diagnosis.—The contagious and fatal character of the malady; the manifestation of buboes, petechiæ, hæmorrhage from the digestive and respiratory organs; the severe fever, with shiverings, ardent heat, giddiness, headache, stupefaction, drowsiness, coma, delirium, muscular twitchings, altered features, injected eyes, insomnia, and great prostration of strength which accompanied it, leave no doubt as to the nature of the malady. The only sign wanting to complete the pathological features of plague is carbuncles; but even these may have existed for what we know about them.

Prognosis.—This was a very fatal outbreak, inasmuch as out of 478 attacks 414 died, and only 64 recovered. At the medical inspection held at Vetlanka, on a population of about 1400, only 10 persons were discovered who bore cicatrices resulting from buboes. The actual population of Vetlanka, when Cabiadis was there, amounted to 1383. The number of deaths which occurred there during the outbreak was 371. The number of those who assert that they were attacked and cured of the epidemic was 81.

Out of this number, those free from all traces of the malady were	52
Those with enlarged lymphatic glands - - -	18
With a running glandular sore - - - -	1
With glandular cicatrices - - - -	10
	81

Epidemic at Vetlanka and its Neighbourhood.

Locality.	Commencement of the outbreak.	Termination of the outbreak.	Population.	Attacks.	Deaths.	Recoveries.	Unaffected.
Vetlanka -	$\frac{14}{26}$ October -	$\frac{13}{25}$ January -	1700	418	357	61	1282
Prischib -	$\frac{5}{17}$ December	$\frac{24}{5}$ December January -	1500	16	16	...	1484
Staritzkoi -	$\frac{15}{27}$ December	$\frac{25}{17}$ January -	2000	9	9	...	1991
Michaelowka -	$\frac{4}{16}$ January -	$\frac{7}{19}$ January -	1200	3	2	1	1197
Selitrenoi -	$\frac{19}{31}$ December	$\frac{26}{7}$ January - February	1600	32	30	2	1568
Totals -	$\frac{14}{26}$ October -	$\frac{26}{7}$ January - February	8000	478	414	64	7522

The Necrological Register, kept by the municipality of Vetlanka, gives a total of 371 deaths, 357 of which fell victims to the epidemic, and 14 died from other causes.

1878—October,	from the	1st	to the	7th,	there died - -	1
„	„	8th	„	14th,	„ - -	0
„	„	15th	„	21st	„ - -	2
„	„	22nd	„	28th	„ - -	3
„	„	29th	„	4th Nov.	„ - -	0
November	„	5th	„	11th	„ „ - -	1
„	„	12th	„	18th	„ „ - -	7
„	„	19th	„	25th	„ „ - -	8
„	„	26th	„	2nd Dec.	„ - -	7
December	„	3rd	„	9th	„ „ - -	56
„	„	10th	„	16th	„ „ - -	169
„	„	17th	„	23rd	„ „ - -	54
„	„	24th	„	30th	„ „ - -	32
„	„	31st	„	6th Jan.	„ - -	19
1879—January	„	7th	„	13th	„ „ - -	12
„	„	14th	„	27th	„ „ - -	0
				Total	- -	371

History.—The outbreak began at Vetlanka on the 14 (26) of October 1878. From the 4th to the 20th of December it assumed a violent type, and raised the death-rate up to thirty-six deaths *per diem*. After the 21st of December it began to subside, and ceased altogether after the 13 (25) of January 1879. From Vetlanka the malady was carried to Prischib, where it broke out on the 5 (17) of December; to Selitrenoi, where it broke out on the 19 (31) of December; and to Michaelowka, where it broke out on the 4 (16) of January, as shown by the preceding tabular statements.

Transmissibility.—All the information hitherto obtained, shows that the malady propagated itself, in the first instance, from the sick to their relatives, and to those who lived with them, or who assisted them during their illness. If, on the one hand, these facts showed its contagious character, on the other hand, evidence is still wanting to prove whether this transmission of the malady was caused by contact with the sick and their clothing, or by breathing an atmosphere impregnated with the deleterious particles emanating from their morbid bodies.

Treatment.—At the commencement of the outbreak, Dr. Döpner believed in the efficacy of large doses of gum arabic mixed with spirits (aquavitæ); mercurial ointment rubbed over the glandular swellings, and a carbolic acid lotion applied, by means of wet compresses, to the open sores. But his hopes were soon disappointed, and the result of this treatment was no better than that of the administration of salicylic acid, of hydrochlorate of ammonia, or of sulphate of quinine. Dr. Cabiadis, therefore, still holds to his convictions that, notwithstanding our acquaintance with the symptoms that characterise plague, we are utterly ignorant of the treatment best suited to its cases.

Incubation.—This generally lasted three days; in some instances it did not go beyond twenty-four hours. There are no data, however, to show the longest period to which it could extend.

Etiology.—The outbreak occurred upon alluvial and damp soil, and in small, low, ill-ventilated habitations, having no latrines, and surrounded by heaps of filth, and amongst a people living in dirt and misery, and feeding on scanty, unwholesome food; breathing, moreover, an air tainted by the numerous "wattagas", or deposits of salted fish, undergoing a slow decomposition. The only sanitary compensation these people can boast of, are the width and straightness of their thoroughfares.

Preventive Measures.—Three cordons were established in

order to isolate the infected places from the healthy part of the Russian territory.

1. The first cordon was put round every locality where plague prevailed, and it prevented persons from entering or quitting that locality until forty-two days had elapsed after the last attack of the malady there. The first cordon that was created, was that put round Vetlanka (viz., on the 20th December (1st January), and the last that was suppressed was that which was put round Selitrenoi, viz., on 28th January (9th February).

2. The second cordon was formed round the infected area, embracing all the smitten localities within its range. Its circumference extended 800 kilomètres, and it was guarded by pickets of soldiers placed at every five kilomètres from each other, and was thus held by 1,600 soldiers. It had four quarantine stations—one on each bank of the Volga to the north, and one on each of its banks to the south. The northern station, on the right bank, was first established at Swetly-Jar, 20th December (1st January). It was afterwards transferred to Vyassowka, 17th February (1st March), and finally to Graczewka. The northern station on the left bank was fixed at Battajefka. Those on the south were established on the 4th (16th) January, one at Zamian, for the right bank, and the other at Petropaulowski for the left bank of the Volga. This cordon was taken off on the 10th (22nd) April.

3. The third, or outermost cordon, was established round the whole province of Astrakhan. It enclosed within its area the two other cordons, and had four stations, or headquarters, one at Sarepta, and one at Besrodnoe, or Vierhno, in the north, and two in the south, at the same stations as those of the inner cordon, viz., at Zamian, and at Petropaulowski. This cordon was established on the 27th December (8th January), and taken off on the 23rd February (7th March). It served to control the functions of the inner cordons, inasmuch as all persons coming from within its area, who could not prove that they had undergone their quarantine at the stations of the middle cordon, were stopped.

None of the delegates visited this cordon.

Hygienic Measures.—Destruction by fire. On Dr. Cabiadis' arrival, he found that only three houses had been burnt at Staritzkoi; later on, five houses were burnt at Prischib, and sixty-seven at Vetlanka, but this was not done until after Cabiadis' departure from those places.

Disinfection.—The houses at Vetlanka were purified by means of sulphur fumes, and by carbolic acid spray; the

latrines, courtyards, and stables were washed with a solution of sulphate of iron; cemeteries were disinfected with lime; clothing was usually disinfected at the quarantine stations with chlorine.

Dr. Cabiadis thinks that the cordons were established *too late*, and that this was probably owing to the conflicting statements given by the medical authorities as to the nature of the outbreaks. He expresses, however, implicit faith in the utility of the cordons, and believes that the most effective of them all was that created by the *panic of the people*, who, to avoid catching the malady, kept away from the sick, and isolated all persons who came into contact with the sick; and did this even before the Government interfered in the matter, and thus succeeded in arresting the extension of the epidemic.

Origin.—Dr. Cabiadis is not inclined to admit a spontaneous evolution of plague in Vetlanka, but thinks that it was imported there from Astrakhan. According to this hypothesis, the next point to determine would be whether it originated in Astrakhan, or was brought there from Resht. Dr. Cabiadis discards the idea of its having been brought from Bagdad, because no direct communication existed between those two places, and the distance between them is very great. Yet, Professor Eichwald seems convinced that plague prevailed amongst the Russian troops in Armenia; that it was brought there by means of the baggage belonging to the regiment of cavalry that came from Bagdad, and that it was thus conveyed to Vetlanka by means of the Cossacks who returned from Kars. In support of his theory, Professor Eichwald quotes the minutes of the Medical Society of Tiflis, in which Dr. Kressnicoffen and some other Russian surgeons state that they noticed amongst the Russian troops in Armenia, a malady exactly similar to the one which appeared in Resht, viz., to bubonic plague. But, on the other hand (observes Cabiadis), Professor Eichwald must also be aware that the majority, as well as the most celebrated military medical officers of the Russian army of the Caucasus, positively deny that plague had shown itself in that army.

Moreover, Colonel Dr. Reutlinger, Superintendent of the Committee for carrying out measures of disinfection at Erzerum, and for collecting all the medical data relating to the Russian campaign in Asia, declared to Professor Zuber and Biesiadecky, that neither at Erzerum nor in any part of the Caucasus had plague shown itself, and that the malady which had prevailed at Erzerum was typhus. Besides this, Dr. Cabiadis quotes the words of Dr. Heyfelder, Chief Phy-

sician to one of the hospitals at Alexandropol during the war, recorded in the tenth number of the French Medical Journal—*Le Mouvement Médical*, published at Paris on the 8th of March 1879, saying: "Je tiens encore à constater que l'épidémie de typhus observée par nous en Asie-Mineure et au Caucase, était bien le typhus pétéchial dans sa forme la plus meurtrière, mais pas autre chose; et n'avait aucun rapport avec l'épidémie d'Astrakhan. Il est probable et presque certain que cette dernière a été introduite dans les villages sur le bas-Volga de Rescht (ville Persane sur la mer Caspienne), et non pas du Caucase." Dr. Cabiadis finally remarks that those who uphold that plague existed in the Russian army of the Caucasus are nearly all civil practitioners, who had neither followed the movements of that army, nor seen its patients; while those who maintain the contrary opinion are chiefly military surgeons who accompanied that army and treated its patients, and who, in consequence, were the most competent to give a correct opinion.

Let us now see what relationship can be established between the date of the return of the Cossacks from the war, and that of the outbreak at Vetlanka.

According to Dr. Döpner, 104 Cossacks started from Vetlanka for the war, and 80 returned to that place. They, therefore, lost only 24 men during a period of more than a twelvemonth's campaign, and they all came back in excellent health. After their return four men were sent away from their companies upon various duties, and returned to Vetlanka at separate periods during the summer.

According to General Attaman Foss, the first company, composed of 32 men, belonging to the second regiment, arrived at Vetlanka on the 22nd of October; and the second company, consisting of 40 men, arrived there on the 3rd of November.

As to their return to Vorpost (Astrakhan), Döpner says that the second company arrived there in two batches, one on the 9th, and the other on the 12th of October; and that the third company arrived there in three batches, one on the 19th, one on the 21st, and the last on the 22nd of October. Döpner, moreover, states that some of these Cossacks were allowed to return to their homes by steamer before the others; that four of them went to Vetlanka, viz., Ivan Kolepof, Ghegor Polakoff, Michael Bellof, and Jacob Bellof. Ivan Kolepof did not fall ill himself, but his wife, Natalie Kolessova, caught (as already told) the malady from her brother, Makar Haritonoff, whom she nursed during his illness, and died of it. The other three Cossacks died in the

beginning of December at the moment the epidemic of Vetlanka was spreading extensively amongst the population.

A leather valise, belonging to a Turkish saddle, was found amongst the effects of the Bellof family, where it was brought either by Michael or Jacob Bellof empty, according to the statement obtained from the Bellof family; but, according to Eichwald, it was supposed to have contained some article brought as a present, and which gave the plague to the family of Bellof, destroying it entirely. This valise was one of the articles shown at the inspection made on the 25th of February, in the open court at Vetlanka.

The outbreak in the Bellova family commenced with Maria Bellova, who had previously nursed her brother Makar Haritonoff, who died of the malady on the 26th of October. She also nursed his wife Prascovia, her own sister Natalie Kolessova, who died on the 9th of November, as well as Vassile Kamass, husband to her sister Onisia, who died on the 27th of November. Finally, she herself fell a victim to the malady, and died on the 2nd of December.

Dr. Cabiadis was unable to ascertain on what day the four Cossacks arrived at Vetlanka; but, as their inspection at Vorpost occurred on the 14th of October, they could not have reached Vetlanka before the 15th of that month, even supposing that the steamer was ready to take them there at once after the inspection. On the other hand, Mavra Pissareff arrived at Vetlanka on the 14th. She had been ill since the 12th, and died on the 17th, and this, without having come into contact either with those Cossacks, or with their things.

With respect to Astrakhan, Dr. Cabiadis observes that in May and June 1877 an outbreak prevailed there, characterised by fever and buboes, similar to the malady which prevailed at Vetlanka in November 1878. Professor Eichwald states that it was not contagious, and that the patients got well. The clinical records of five cases of this outbreak were forwarded to St. Petersburg, but Dr. Cabiadis was not able to see them. It is well known, however, that some of the members of the Medical Council of St. Petersburg declared that it was *the plague*, and in consequence of this declaration, energetic sanitary measures were adopted. Dr. Cabiadis moreover remarks that even the Vetlanka outbreak was not at first recognised as one of plague, but only as a fever of an intermittent or remittent type; and this error arose in consequence of the doctors not having remarked its *contagious properties*. For the same reason, no doubt, Drs. Nicolski, Gregorieff, and Golubef, and Colonel Pelanoff telegraphed to General Foss, on the 28th December, that plague

did not exist at Vetlanka, but that the outbreak there was one of croupy-pneumonia, and that its fatal character was owing to the detestable hygienic condition of the population, and that no quarantine measures were therefore necessary.

Dr. Döpner, when he went to Vetlanka on the 18th November, found there an outbreak of remittent fever with buboes; not contagious, but exactly similar to the outbreak he had seen in Astrakhan. On his second visit to Vetlanka, on the 5th December, he acknowledges that he had been mistaken with regard to its contagious character, for he now found that it was, on the contrary, very contagious; he therefore called for quarantine restrictions. There were other doctors who also held a similar opinion.

At the commencement of the outbreak, that is, at the period when buboes freely manifested themselves, the mortality was not great, and the patients usually got well; but later on, when the epidemic had spread over the people, it became very fatal, and destroyed those smitten by it in a few days, or even in a few hours, and before buboes had time to appear. This circumstance has been noted in all outbreaks of the plague.

The outbreak at Astrakhan never assumed a fatal character. This feature was remarked also, on several occasions, in Mesopotamia during the year 1876, viz., at Musseyeb, Kerbela, Bassorah, etc., the malady limiting itself to simple glandular engorgements, sometimes with and sometimes without fever. These glandular swellings generally subsided, and disappeared without suppurating, though occasionally they did so, and they never showed any indication of a contagious character.

When an epidemic outbreak is marked by a contagious and fatal tendency, these strengthen the probability of its pestilential nature; the absence, however, of such characters does not necessarily exclude the possibility of that outbreak being the plague. Dr. Cabiadis, therefore, supposes that the Astrakhan outbreak was a mild form of *bubonic plague*. Assuming this as a fact, the question now presents itself: Did this plague originate spontaneously at Astrakhan, or was it conveyed there from Resht?

The plague was known to exist at Resht from the month of March 1877 until October of that same year; and, according to some authorities, even up to the summer of 1878. Frequent and uninterrupted communication existed between Resht and Astrakhan, and no quarantine measures were established at Astrakhan, previous to the 17th (29th) of May, that is to say, nearly two months and a half after the period

at which the plague had declared itself at Resht; the intercourse between these two cities remaining all the while absolutely free. This fact leads Dr. Cabiadis to suppose that the malady of Astrakhan was derived from Resht.

The following are the conclusions of Dr. Cabiadis' long, but interesting, report:—

1. The Vetlanka outbreak was undoubtedly one of *bubonic plague.*
2. It probably derived its origin from Resht.
3. The outbreak commenced at Vetlanka, on the 17th (29th) October 1878; and ended at Selitrenoi, on 28th January (9th February) 1879.
4. From Vetlanka it spread to Prischib, Staritzkoi, Selitrenoi, and Michaelowka.
5. In May and June 1877, a suspicious outbreak prevailed at Astrakhan, which was in all probability an outbreak of bubonic plague of a mild character.
6. The outbreak at Vetlanka, Prischib, Staritzkoi, Selitrenoi, and Michaelowka had evidently a contagious and fatal tendency.
7. In some places the malady was restricted to a few cases, and was arrested from the very commencement by means of quarantine measures, while in other places it assumed an epidemic character.
8. The *Cordons-sanitaires* established against this outbreak proved extremely efficacious.
9. In a population numbering about 8000 souls, there occurred 478 attacks, and 414 deaths from the epidemic.

Tzaritzyn, 10 (22) *April,* 1879.

ON CERTAIN APPEARANCES OF CHOLERA IN THE COUNTRIES LYING BETWEEN EUROPE AND INDIA SINCE THE YEAR 1874.

By J. NETTEN RADCLIFFE.

(*Read: November 3rd*, 1880.)

THE great diffusion of cholera in Europe which began in 1865, and acquired new vigour in 1869, came apparently to an end in 1874. The disappearance of the disease on the Continent in the course of that year is, however, a matter of assumption rather than of ascertained fact. In 1873 cholera was extensively prevalent in European Russia, Poland, and Central Europe, and it spread over a considerable portion of Western, and some parts of Southern Europe, including Bosnia, the Austrian provinces on the eastern coast of the Adriatic, several parts of northern Italy and of the Italian coast on the Mediterranean. The disease is reported to have ceased in Russia in January 1874; it was seriously prevalent in parts of Central Europe in the summer of that year, and there is reason to suspect that there was a renewed prevalence in Poland; but as the year advanced into the autumn the cessation of all news as to the existence of the disease, and the absence of any information as to its then existence in any part of Europe, during the sittings of the International Sanitary Conference in the month of July 1874, appeared to justify the assumption that the renewed diffusion, beginning in 1869, had come entirely to an end before the close of 1874. At this time, moreover, there was reasonable ground for the belief that the countries lying between Europe and India were free from cholera, and that while Europe had thus become relieved from a present evil, there were no indications of a recurrence of that evil in the near future.

Epidemiologists were taken by surprise then, when towards the end of May 1875, and at the beginning of June, it became known that cholera existed in northern Syria. At first, doubts were entertained as to the actual nature of the disease; but these were presently set at rest by the concurrent testimony of several competent medical men, familiar

with cholera, and who, from actual observation of the malady, concluded they were dealing with that disease.

According to the official data, cholera broke out at Hama in northern Syria in the course of March 1875, and (spreading thence, as it were from a centre) in the course of the next seven or eight months extended over a tract of country including Taurus on the north, Orfah, Damascus, and the Hauran on the east, Safed (in Palestine) on the south, and many of the coast towns from Alexandretta to Tyre on the west.

Hama, the town in which the disease first appeared, is situated on the river Orontes, in a wide plain which extends between Aleppo and Damascus, and it lies about half-way between the two cities. The river in this part of its course passes through a deep gorge, the town occupying the steep banks on either side, and immense water-wheels on the edge of the stream serve the purpose of raising water for the use of the inhabitants and for irrigating the numerous luxuriant gardens. Above the town the water of the stream, although coloured with the ferruginous clay which constitutes the soil of the locality, is reported to be good; but below the town (and within its limits I suspect) it is fouled with the liquid refuse which finds its way into the river from the houses on both sides. The wealthy classes occupy large airy houses in good positions; but the greater part of the population is badly lodged, living in narrow close alleys. Their houses, constructed of sun-dried bricks, are small, one storey in height, and arranged in the following manner. Round a little court, opening into the street, are placed two or three small rooms, each having a narrow entrance, and a little window alongside of it. Every room is usually occupied by an entire family, which, in some instances, is composed of three generations. Few of these houses possess latrines; hence the adjacent street serves as the common receptacle for excrement and house sweepings. Wood and charcoal are very scarce at Hama. The inhabitants use, instead, camel and cow-dung as fuel, and this serves for both cooking and warmth. The houses are always surrounded by filth of every description.

The population of Hama is variously estimated at from 30,000 to 40,000, and Mr. Vice-Consul Jago styles it the Manchester of Syria, owing to the extent and variety of its cotton, silk, wool, and goats' hair manufactures. Its sheets, napkins, table-cloths, towels, and under-clothing are in great requisition in Eastern households throughout Syria, and also in Turkey; and it is an important mart for the supply of camp and camel-furniture, and for clothing to the Bedouin

tribes. Tripoli and Tortosa, distant each about seventy miles (a four days' journey in the existing state of the roads) are the outports of the town.

The first case of cholera, to which official attention was first especially directed, occurred in the military hospital on the 22nd of March 1875. The hospital, at the time, was crowded with recruits, recently arrived from Albania, in an exhausted condition from the fatigue of the journey, and sickness. A fortnight after the attack referred to, the disease appeared among the civil population of the town, in which it appears to have prevailed until the month of July, giving rise to considerable mortality. Aleppo was attacked in May; Antioch, Damascus, the Hauran, and Homs in June; Beyrout, Latakia, Sidon, and the Lebanon in July; Tripoli, Djebail, Akkar, Deir, Aintab, Killis in August; Tyre, Orfa, Beredjik, Marash, Taurus (Hisna Mansour and Abbas), Tiberias, and other places in September; Safed (in Palestine) in October; and Nazaret and Kaifa in November. The total deaths from cholera recorded in these several outbreaks (where deaths were recorded at all, and in several instances the fact of prevalence was merely stated) exceeded 9,000; and there is no pretension that this number represented the actual loss of life.

On the news of the appearance of cholera at Hama reaching Constantinople, the Ottoman General Board of Health telegraphed instructions to their sanitary physician at Beyrout, Dr. Pestalozza, to proceed to the scene of the outbreak and make inquiry as to the actual nature of the disease reported to be cholera and into its origin.

Dr. Pestalozza furnished a careful report of the results of his inquiry in June to the Board, and an admirable summary* of this report, prepared by Dr. S. Dickson, the British representative at the Board, was transmitted to the Foreign Office. I regret that the necessity of having regard to time prevents me reading this summary to the Society *in extenso*, but I have made use of it freely in what relates to Hama.

Dr. Pestalozza, as previously intimated, confirmed the original reports of the nature of the disease which had appeared at Hama, but he was unable to discover any source exterior to the town, from which it could have been derived. There

* I use the word summary, following, in effect, Dr. Dickson's own phraseology, but, in reality, Dr. Dickson's "summaries", as the Society has previously had occasion to observe, are independent papers, based upon the information before him, but "informed" (if I may use the word in this sense) by his rare knowledge of the subjects he has to deal with, and his unrivalled acquaintance with the sources from which the knowledge is derived in Turkey, and their value.

were remote possibilities that the disease might have been introduced into Syria by troops recently returned from Yemen, by a caravan of Persian pilgrims from Bagdad which had visited Hama shortly before the outbreak, and by the Albanian recruits previously referred to, and among whom the disease was first recognised. But there is no knowledge of any cholera having existed in any of the Turkish possessions on the Red Sea, when the troops left there, or of any choleraic affection having occurred among them on their route to Hama, by way of Beyrout. In like manner no choleraic affection could be ascertained to have occurred among the individuals composing the Persian caravan; and it is tolerably certain that at the time of their leaving Bagdad no cholera existed in the valleys of the Tigris and Euphrates from their head-waters to the Persian Gulf. No suspicion of cholera appears, moreover, to have been entertained of the existence of the disease in Albania, or among the Albanian recruits on their route to Hama. In short, Dr. Pestalozza's efforts to discover a source of introduction of cholera into Hama failed, and he came to the conclusion that the disease had originated on the spot. This conclusion was very generally adopted by medical men in Syria and Turkey, and still continues to be held. It has been debated even whether the outbreak was not determined by a revivification of cholera contagion which had remained inactive in the town since 1865, in which year Hama suffered severely from the disease as it had also done in 1848.

But reviewing the whole circumstances of the diffusion of cholera westwards and northwards of India from 1865 to 1874, I have elsewhere had occasion to express the opinion that "Wherever it has been possible to follow the diffusion in detail, its history has been that of a dissemination by human intercourse. Wherever the details of that diffusion have failed, I am unaware of any other suggestion which would explain the successive chronological phenomena."* Now it seemed to me at the time (and it still seems) highly improbable that one of the most marked characteristics of the phenomena of the diffusion of 1865-74 should suddenly disappear in respect to an outbreak which was obviously one of the latest manifestations of that diffusion; and, further, that Dr. Pestalozza had simply failed to discover the source of the outbreak (which was the fact) either

* See "Report on the Diffusion of Cholera, and its Prevalence in Europe, during the Ten Years 1865-74," published in the *Reports of the Medical Officer of the Privy Council and Local Government Board.* New Series, No. V, 1875.

from not having got hold of the right clue, or from having had to conduct the inquiry at a time when it was found impracticable to recover the clue. There are not wanting reasons to justify this conclusion other than an inference from the failure of the inquiry in this respect. And, first, it is doubtful whether the recognition of cholera in Hama in March really marks the beginning of the disease there. A military medical man, named Giovanni, speaking to Dr. Pestalozza of the unusual character observed in certain cases of disease in Hama, the autumn and winter preceding the recognition of cholera, observed that had cholera been prevalent it existed in the winter at Hama. If this were correct, the beginning of the outbreak would be thrown back to the later months of 1874, and a search for its origin on the assumption that it commenced in March would almost necessarily prove futile.

I have referred in a previous paragraph to the prevalence of cholera in Bosnia, and on the Adriatic littoral in 1873. I am ignorant of the details and duration of that prevalence; but it is certainly a curious coincidence that its latest reported prevalence in southern Europe in 1873 should refer to the districts, or, if you will, countries, adjoining those from which the recruits were drawn among whom the disease was first recognised in Syria in 1875. Moreover, is it so sure that these parts of Europe, and the parts with which Hama would be in communication through its outports, Tripoli and Tortosa, were so free from cholera as is assumed? That there are still, probably, facts of the history of cholera on the continent in 1874 to be ascertained is made abundantly evident by what I am now going to state, and possibly some of the facts, when described, may afford the missing clue to the outbreak at Hama.

In the number of the *Annales d'Hygiène Publique* for the third quarter of 1876, a paper appeared from the pen of Dr. Lacassagne, one of the professors at Val-de-Grace, respecting an outbreak of cholera in Algeria in 1873-74. This paper made known, for the first time, that an outbreak of cholera, which had begun in October 1873, and continued to February 1874, had broken out in the military circle of El Melia, in the eastern part of the province of Constantine. The outbreak affected several villages and encampments, and caused 257 deaths. Until the appearance of Dr. Lacassagne's account of this outbreak no suspicion of the existence of cholera in Algeria at the time in question appears to have existed among the powers of the Mediterranean littoral, and none of the measures customarily adopted among them for

protection against cholera were in consequence put in action. It must be added that none of the powers came to any harm from the want of them. The reporter of this outbreak, as afterwards the reporter of the Syrian outbreak, came to the conclusion that the disease had originated on the spot; but the reporter of the Academy of Medicine, commenting afterwards on this conclusion, pointed out that there were probabilities of the infection having been introduced from France, which at the time the investigators of the El Melia outbreak were not aware of. Want of knowledge of the languages current on the eastern littoral of the Adriatic, and also of time to cope with an inquiry, peculiarly difficult in itself, but infinitely more difficult if attempted without this knowledge, has prevented me from attempting to follow the history of the prevalence of cholera on that littoral in 1873. But I think it highly probable that a clue will be found there, at some future period, to the outbreak in Syria—a clue similar to that which the reporter of the Academy of Medicine gives us to the outbreak in Algeria.

The outbreak of 1875 in Syria occurred at the time of one of the great epidemic-extensions of cholera in India, and proceeded, it might be said, concurrently with the progress of that epidemic outside the limits of Bengal. Dr. Bryden, indeed, writing of the first recognised case of the Syrian outbreak, regards it as "a true homologue of our cholera of the same day [that is to say, of a case of cholera which, he says in a previous sentence, appeared in Nasik "the very same day", 22nd March, 1873],* and that the revitalisation of the same cholera on the 20th July was one and the same phenomenon in Syria, and in northern, western, and southern India. In my estimate it was one and the same cholera renewing its epidemic manifestions, which constrained Dr. Vost, of Briont, to write, dating his letter 3rd August, impelled Dr. Townsend to write on 4th August reporting the invasion of his province, the Madras Government to telegraph to Simla the advent of the new epidemic, and the Government of India to write to ask what this cholera meant, and what the end was likely to be."† In 1876 this epidemic extension involved Kashmir, parts of Afghanistan, and Beluchistan. In the summer the disease was seriously prevalent in Seistan and the valley of the Helmund, and at the close of the year it was reported on the Persian frontier, and rumours existed in Persia of its presence in Herat.

* *Reports*, 1876, and *The Cholera History of* 1875-6. Calcutta, 1878. P. 278. "One death occurred in the town of Nasik between the 22nd and the 31st of March."

† *Reports*, 1876, p. 312.

While cholera had thus been spreading to the north-west, it had been extending also to the countries east of India, and into the islands of the eastern Archipelago (Malasia). The history of this extension has yet to be written. This has not yet been feasible, but recent contributions to the subject, particularly with regard to the prevalence of the disease in Japan and in China, will probably induce some epidemiologist to attempt the task.

The presence of cholera in Afghanistan on the Persian frontier, near the close of 1876, the severity of the disease as there observed, with the phenomena attending its extension, gave rise to serious fears at Teheran that another invasion of Persia by the malady was about to take place. This, however, did not happen, and the north-western extension of cholera in the direction of the Persian frontier seems to have come to an end with a disastrous outbreak among a body of workmen engaged in building a dyke upon the river Helmund.

It was not until the autumn of 1877 that any further news of any westward extension of cholera was received. On the 23rd August of that year the telegraph flashed the information from Alexandria that a French troopship from Saigon had passed Aden, bound for Suez, with cholera on board. This vessel, the *Correze*, had left Saigon, bound for Toulon, on the 19th July. She carried 770 troops, and the day after sailing cholera appeared among them—sixty cases, thirty-two of which proved fatal, occurring from that date (the 20th of July) to the 16th August. The outbreak ended two days before the vessel touched at Aden, and the last patient had left hospital before she reached Suez, on the 24th August. There would appear to have been much sickness on board this ship, as well as cholera, for, on being sent by the Egyptian authorities to Tor, to undergo a ten days' quarantine, she landed there 130 invalids, suffering, as the report runs, from "ordinary maladies".

The outbreak of cholera on board the *Correze*, although doubtless it had no direct relation with what followed, proved to be the forerunner of an outbreak of the disease in the Hejaz during the month of December following.

The Mecca pilgrimage which occurred in 1877* fell in the month of December. The numbers of pilgrims assembled at

* The particulars as to this pilgrimage and the outbreak of cholera among the pilgrims, which occurred after the termination of the rites, are mainly taken from a report of the Egyptian Board of Health, namely, *Exposé des mésures prises en Egypte contre l'Epidémie Cholerique du Hedjaz de* 1877-78.

Arafat on the great day of the rites (14th December) is estimated at 100,000, of whom the larger proportion came from the far east, one fifth only having come from Egypt, or journeyed to the Hejaz by way of Egypt.* The pilgrimage ended on the 16th December, and, as customary, the dispersion of the pilgrims began at once. Concurrently with the close of the pilgrimage the weather broke, torrential rains fell, and the temperature was much lowered. At this time, and for several days afterwards, there was no suspicion of any unusual sickness among the pilgrims, and the Ottoman Medico-Sanitary Commission, when reporting the termination of the pilgrimage, stated that during the four days of the rites no exceptional sickness had been observed, that the deaths were comparatively few, and all from ordinary causes, and that the only malady which had called for remark was small-pox among the Javanese pilgrims. On the 23rd December a case of cholera was recognised in Mecca, and the next day not less than 102 deaths from this disease were recorded. About the same time the disease appeared among several of the pilgrim caravans which had already left Mecca, and on the 25th December eight fatal cases occurred in Jidda.

From the 23rd December 1877, to the 11th January 1878, the number of deaths from cholera recorded in Mecca was 785; in Jidda, from the 25th December to the 8th January, the number was 186. The Syrian caravan, which appears to have numbered about 3,000 pilgrims, and which left Mecca on the 19th December, had, between that city and Medina, which latter city was reached on the 6th January, 169 deaths from cholera. The Bagdad caravan, numbering about 2,500, had also on the same route 10 deaths from cholera. The Egyptian caravan, which numbered only 830, did not suffer from the disease. At Medina, cholera was first noticed on the 31st December, and from that date to the 12th January the disease would appear to have caused 163 deaths. Cholera in this city seems to have occurred almost wholly, if not wholly, among the pilgrims who flocked into the city coming from Mecca. The inhabitants who were attacked, it is stated, were themselves pilgrims, returning from Mecca.

When the Syrian and Bagdad caravans left Medina they were reported to be entirely free from the disease, but this

* Of the pilgrims who had arrived at Jeddah up to the 1st December 1877, the Javanese numbered 8,086, the Moors and Tunisians 6,732, Egyptians 4,634, Indians 4,565. Turks 3,271, Persians 1,757, Arabs from Yemen 751, pilgrims from Massowah (Abyssinians, Nubians, etc.) 437 = 30,230.

was not the case with the numerous caravans which took the route for Yembo. None of the caravans returning by this latter route had attached to them a medical man, and no medical man was stationed at Yembo. Hence, a medical history of what occurred on the journey is wanting. From first to last, the number of pilgrims who congregated at the port, waiting embarkation, is estimated to have been not less than 10,000. No provision had been made for regulating the encampments there, or for supplementing the supplies of the place. The pilgrims underwent great privations, not only from insufficiency of food, but also from an exceptional severity and tempestuousness of the season, and the hygienic condition of the town and encampments became most revolting. It is stated that no cases of cholera occurred among the pilgrims while at Yembo, but that there was much choleraic diarrhœa, and twenty-nine deaths were reported from this cause from the 18th January to the 5th February. Cholera was reported among pilgrims travelling southwards by way of the Assyr country, and the disease showed itself at Confodah among two battalions of troops who had marched there from Yemen, and had contracted the disease in a village on the route previously infected by pilgrims returning from Mecca. The malady was confined to the troops, of whom it killed twenty-nine, no extension taking place among the civil population. Further south on the coast, cholera appeared among a body of returning pilgrims who had been placed in quarantine in the lazaret of Hodeida. The number under detention at the time was 1,300, but of these a body of 300 appears alone to have been attacked. Among this limited section there were eighty cases and forty-nine deaths. The disease was confined to the lazaret and to the body of pilgrims within it. North of Yembo a few cases of cholera occurred among the returning pilgrims kept in quarantine at the Egyptian station, Tor, on the east coast of the Gulf of Suez. There 14,100 pilgrims underwent quarantine from the 30th December 1877, to the 28th March 1878; and among this number five deaths from cholera occurred between the 30th December, the day on which the first body of pilgrims was landed at the station, and the 8th January, all among pilgrims landed from the same ship. A sixth death from the disease occurred on the 30th January. After this date no case of, or death from, cholera appears to have been recorded in Western Arabia or within Egyptian territory.

Of the pilgrims who had to pass through the Suez Canal on their return, the first body who had completed their quarantine entered the canal on the 29th January 1877, and

the last on the 30th March. The whole period covered by the return of the pilgrims along the sea route to Suez, with the quarantine to which they were subjected, was three months. Of the 14,100 pilgrims who underwent quarantine at Tor, 7177 were from destinations beyond the maritime canal, and were sent forwards by that route when they had purged their quarantine.

In May 1878, the fleet of ships employed in conveying the British Indian Contingent force from Bombay to Malta, passed through the Suez Canal. Two of these ships had had cholera on board during the voyage, but one of them was granted at Suez free *pratique*, twenty days having elapsed between the occurrence of the last case of cholera and her arrival there; and the other, a less period of freedom having taken place between the last death on board and her arrival (twelve days), was sent through the canal in quarantine.

Not long after the contingent had been landed at Malta a rumour spread throughout the Levant that cholera had broken out in the island. The rumour was unfounded, but in the course of August a serious prevalence of cholera in the interior of Morocco was officially reported by the various foreign representatives resident in that country, and afterwards it was made known that the disease had spread from the interior into the principal seaports of the country—Mogador, Rabat, El Araisch, Tangiers, Melita—and had extended to the Algerian frontier at Oudja.

Such is a very brief summary of the manifestations of cholera in Arabia and North-Western Africa in 1877-78. Two questions of cardinal epidemiological interest arise out of them, and to these I propose to devote a few moments' consideration. The first of these questions relates to the source of the outbreak in the Hejaz; the second to the nature of the disease in Morocco.

1. The outbreak of cholera in Mecca in December 1877, as I read the facts, took every one concerned in the medico-sanitary welfare of the city, and its seaport Jedda, by surprise. Nothing appears to have occurred at the time of landing of the pilgrims at Jedda, and during their assembly from various places at Mecca, or in the course of the rites, to lead the medico-sanitary authorities to suspect the presence of the infection of cholera, or any tendency to cholera, among the multitude. Failing any information or suspicion of previous infection, the conclusion was arrived at that the outbreak was of local origin, determined by the exceptional local meteorological conditions which obtained concurrently with

the close of the pilgrimage. In other words, unable to discover a probable or possible source of infection, it was concluded that infection had no part in the outbreak. The conclusion may be right, but, under the circumstances, the ascertained facts can scarcely be accepted as establishing the autochthonic origin of infective cholera in Arabia in 1877. And first, so far as I know, this has not been the source of the previous outbreaks of cholera in Arabia which have admitted of closest study, and the probability is therefore against a local origin. Again, the conclusion appears to have been formed in ignorance of the state of the countries in Central and Southern Arabia (except only such as came under, or were immediately contiguous to those under, Turkish rule), with reference to the presence of cholera; and I have shown elsewhere, in respect to the occurrence of cholera in Hadramaut in 1865, that without this knowledge conclusions as to cholera at Mecca may be very fallacious. Further, the sources of information at the command of the Ottoman medico-sanitary staff at Mecca and elsewhere, and of the foreign representatives at Jedda, while sufficient to deal with the more obvious indications of infection, are not such as to give much confidence in results where less obvious indications are concerned. Still further, the chronological relations of the outbreak to the extensions of cholera beyond the north-western frontier of India in 1876, and in the Eastern Archipelago in 1877, are to be had in mind, and, assuming that the outbreak followed the course most ordinarily observed in westward extensions of cholera, it is probable (putting aside the question of accidental but unobserved introduction of the disease in the tract of the pilgrims coming from the far East) that cholera existed in some parts of Central and Southern Arabia, from which pilgrims visited Mecca, before the appearance of the disease in that city.

2. A serious doubt rests upon the actual nature of the disease which prevailed in Morocco. On the news reaching Tangiers, in August 1878, that "a strange and fatal disease had appeared at Fez and Mequinez, which had caused considerable mortality, and which it was suspected might possibly be cholera,"* the Board of Health despatched a Spanish physician, Dr. Tadeo Martinez, to Mequinez, with instructions to investigate and report to the Board concerning the nature of the disease. Dr. Martinez, as the result of personal observation, came to the conclusion that the disease

* Mr. Horace P. White, Chargé d'affaires and Consul-General, Tangiers, 21st August 1878.

was "endemic cholera morbus Asiatica," and he attributed the malady to "the scarcity and dearness of the bread-stuffs, the increased consumption of fruit, and the unwholesome quality of the water, combined with the state of filth which exists in the town, particularly in the Jewish quarter."* The report was not such as to give much confidence in the accuracy of the diagnosis, or rather was such as to leave the question of the actual nature of the disease unsolved; and presently, when instances of the malady began to be observed in the seaport towns, the opinion that the disease was, in any sense, a form of Asiatic cholera was altogether discarded by the European medical men residing there, and who appear to have had sufficient opportunities for observing it. The medical officers to the Boards of Health of Tangier, Mazagan, and Mogador, Drs. Migueres, Ovilo, and Martinez (apparently not the Dr. Martinez sent on the first summons of the outbreak to Fez, viz., Dr. Tadeo Martinez) concurred in the opinion that the disease was not cholera. Dr. Allard, a French physician at Mazagan, reported to the Minister of France at Tangier to the same effect; Dr. C. Ollive, of Mogador, reported also in a like sense to the French Consul there; and the Austrian Consul at Tangier, Dr. Schmidt, a medical graduate of the University of Glasgow, expressed the same opinion. A good deal of enteric and fatal choleraic disease existed, particularly among young children, and Dr. Schmidt observes of these diseases as observed in Tangiers—

"If the sickness which exists actually in Tangiers, and without doubt in an epidemic form, was cholera, we should have to call it the cholera of children; for nine-tenths of those attacked with diarrhœa and vomiting are from three to twelve years of age, while the remainder are persons of advanced age and feeble state of health, and persons who exist without the means of existence (if I may be permitted to make use of such an expression). Very different is the action of true cholera. . . . . If it be asked, then, what kind of sickness is that which now exists in Tangiers? my opinion is that it is not one well-defined malady only, but rather a complexity of affections of the digestive organs—affections which in some individuals develop into dysentery, in others become catarrhal or inflammatory gastro-enteritis, and in others pass into gastric and abdominal fevers. Now it is certain that the symptoms which characterise dysentery and enteritis, etc., are very well marked and not easily confounded. This is the case when they come to take an epidemic and malignant character and extension, as now

* Mr. Horace P. White, August 21st, 1878.

happens at Tangiers, and as happened here some weeks since, and as I believe has happened for some four or five months past in the different ports of Morocco; then they cannot but exhibit a great analogy even with the marked symptoms of cholera, etc."*

Dr. Schmidt referred also to the absence of diarrhœa, such as usually accompanies an epidemic of cholera.

The other physicians whose names have been mentioned expressed opinions to the same effect; and it may be here observed that an outbreak of malignant dysentery, which occurred among the Sultan's troops the year following, was regarded as the same disease as that which had prevailed largely among the civil community the previous year.

There was a general concurrence of opinion among the foreign representatives in Morocco and their medical advisers that the suspected choleraic and actual choleraic affections had been determined by the dreadful privations to which the poorer classes of the people had been subjected in 1878, and more particularly to the use of a flour prepared "from the root of the *arum*, which it is stated, when not properly prepared, acts as a deadly poison, producing symptoms similar to those of cholera."

Dr. Tadeo Martinez reported that the outbreak in the Jewish quarter at Mequinez (and apparently the commencement of the outbreak in the town) began on the 21st July 1878. This date is of considerable interest when considered in relation to the foregoing view of the nature of the disease, and in connection with the following account given by Mr. Consul Drummond Hay of the state of Morocco in 1878. He writes:—

"In my trade report for 1877 I mentioned that swarms of locusts had appeared in the country, and devoured the young crops, which were then most promising. These flights of locusts were not confined only to Mogador, but extended over the large tracts of country ranging between Soos, Morocco city, and the coast as far north as Mazagan.

"Farmers and landowners in 1877 suffered considerably from the visitations of these destructive insects, and the agricultural prospects for 1878 were looked forward to by the natives with anxious hopes that they would be repaid for their misfortunes by a good year.

"The ploughing season, however, passed by without any rain falling, excepting a few showers, which were barely sufficient to moisten the earth. When spring arrived, the

* Translation of a letter communicated by Mr. Drummond Hay, Tangiers, December 5th, 1878.

few crops of corn that had been sown were scorched by the sun, and were unable to throw out the ear.

"In ordinary years, after a seasonable winter, the mountains and plains around Morocco are covered with verdure and wild flowers, but in the spring of last year hardly a blade of grass was to be found in the southern provinces. The mortality amongst cattle therefore commenced at an early stage of the drought. In wooded parts camels and goats contrived to exist by browsing amongst the parched trees and shrubs, but by the end of the summer even those hardy and long-suffering animals began to die off by thousands.

"As early as the month of April, fugitives from the famine-stricken provinces appeared on the coast seeking relief at the ports. The number of refugees continued to increase daily, and by the month of June the towns of Mogador and Saffi were crowded with starving and emaciated creatures. Many of those who arrived at the ports quite in the early stage of the drought, before being reduced to a state of starvation, found employment sufficient to keep body and soul together, but the later arrivals were all in the last stages of starvation, having the appearance more of walking skeletons than of human beings, and either immediately succumbed to the fatigues of the journey, or fell easy victims to the various diseases which were rife amongst them, such as small-pox, dysentery, cholera, and typhus.

"The sanitary condition of the towns which were afflicted with large numbers of the refugees, such as Mogador and Saffi, suffering severely from their presence, and each of the diseases which I have enumerated above became an epidemic, carrying off a large percentage of the native population and many Europeans. The governors and sheikhs of the provinces in the interior did not relax their arbitrary and tyrannical exaction of tithes and taxes; thus thousands of poor peasants who might have managed to struggle through the distress were forced to leave their homes and flee to the towns, full of pestilence and disease, where most of them found their graves.

"At Morocco city and the ports of Mogador and Saffi caravanserais were provided by the Moorish Government for housing the homeless strangers, and at the east ports soup kitchens were established for their relief, by order of the Sultan. The dead received a decent burial at the expense of the Moorish Government, and thus bodies were not left to putrify in the streets as is usually the case in the East during times of famine and epidemics. Hundreds of unfor-

tunate fugitives were found dead on the high roads in the interior, half devoured by jackals and carrion-eating birds, but these sad sights were seldom to be seen near the villages and inhabited parts of the country.

* * * *

"It would be impossible to give an accurate return of the number of people that have perished in consequence of the famine, owing to the want of statistics, but from a rough calculation of the number of inhabitants formerly known to exist in the now nearly depopulated districts I cannot estimate the deaths at less than 200,000."

To this point, the data which had reached this country tended to the conclusion that the fatal choleraic disease which had been prevalent in Morocco in 1878 was not true cholera. But in the course of 1879 Dr. Linares, a French military medical officer, attached to a military mission despatched from Algeria to Oudjda, a town within the territory of Morocco, a short distance from the Algerian frontier, printed privately a report on this choleraic epidemic. I am indebted to Dr. Vallin, Professor of Hygiene at the School of Val de Grace, for an opportunity of reading this report, he having courteously placed his own copy at the service of the medical department of the Local Government Board. Dr. Linares, whilst at Oudjda, had an opportunity of seeing three cases of the disease, and he entertains no doubt that they were cases of true Asiatic cholera. He instituted a diligent inquiry into the whole circumstances of the outbreak, and (influenced by his view as to its nature) he has drawn up a somewhat detailed account of its development and movements.

I restrict myself to Dr. Linares' account of the three cases of the disease he himself saw. It is as follows:—On the 1st Sept. two soldiers of the garrison of Aïoun-Sidi-Mellouk, Mouley Ahmed (a captain) and El Madjoub (a sergeant-major), arrived at Aïoun, Oudjda. The heat at the time was great, the weather stormy, and the sky overcast. At Aioun the epidemic was in full development. These soldiers took up their quarters, both in one chamber, on the ground floor of a dwelling level with the interior court and back from the street. A little straw spread on the floor, and over which a coverlet was thrown, formed their bed. Hardly had they installed themselves in this room than El Madjoub fell ill. He was attacked with vomitings, purging, and frequent serous stools, cramp in the limbs, tendency to algidity; in a word, cholera. He died the next day (the 2nd of September).

The day of El Madjoub's death, Mouley Ahmed was attacked, and presented symptoms similar to those which had been observed in his companion. When first seen he was sitting upon his coverlet, his shoulders resting against the wall; the face was slightly cyanosed, and having an expression of agony. The eyes were hollow; the feet and the hands nearly cold. Vomiting was frequent, the stools serous, and thirst was extreme. He complained of cramps in the calves of his legs, and spoke in a low, scarcely audible voice. The next morning, the 3rd of September, Dr. Linares was surprised to find Mouley Ahmed living. The vomiting and the stools had become less frequent, but the character of the face remained unaltered. The stupor (now first mentioned) and the tendency to cooling persisted. 4th of September: the patient remained nearly in the same state. Vomiting much less frequent, but the purging continues undiminished; the stupor also continues. 5th of September: the patient's state is sensibly better. 6th of September: the patient was found in the morning in a very grave condition. The symptoms observed at the beginning had recurred, and death occurred in the course of the evening.

On the morning following the death of Mouley Ahmed (7th of September) a soldier of the Oudjda garrison, who alone had attended to him, was attacked with cholera. "Contagion", observes Dr. Linares, "was here very evident." The symptoms presented by this case, although less grave than in the two previous cases, were very significant. 8th of September: the patient's state continued nearly unchanged. Vomiting and purging were frequent; stupor marked. 9th of September: the patient a little better. 10th of September: the patient still better. The vomiting continues, but the purging less; general feebleness very great. 11th of September: the improvement continues, but the adynamia persists. 12th of September: state unaltered. 13th of September: the patient worse in every respect; the condition very grave. The vomiting reappeared during the night, and the choleraic symptoms manifested themselves anew. The adynamia is extreme. The patient answers questions with difficulty. Informed that the diarrhœa had returned, and that the stools were sanguinolent; Dr. Linares ascertained that the patient had suffered from very considerable hæmorrhage from the intestines. The pulse was filiform, the stools involuntary, the extremities cold, and death occurred in the course of the evening.

No other case took place, either among the soldiers stationed at, or among the civil population of, Oudjda.

I leave it to the members of the Society to judge how far these cases, which are given as Dr. Linares gives them, support his contention that true cholera existed in Morocco in 1878. If such were the case there is no need to seek its origin, as he is disposed to seek, in the revivification of a persisting focus of the disease left by the epidemic of 1867, after the fashion of the analogous phenomena observed in Galicia and Hungary from 1848 to 1852, according to Tholozan.

The facts I have given respecting the Hedjaz outbreak would perhaps justify the more probable inference that there had been a reintroduction of cholera into Morocco by the Moorish pilgrims returning from Mecca in the course of the first three months of 1878; in other words, that the epidemic of cholera of 1878 in Morocco was an offshoot of the outbreak of 1877-78 in the Hedjaz. But a conclusion to this effect would have a particularly awkward bearing upon the question as to the real influence of the quarantine maintained by the Egyptian Government against the Hedjaz during the continuance of the outbreak there and the return of the pilgrims. The measures then adopted by the Egyptian Local Board of Health have been spoken of as a crucial example of well-devised and maintained quarantine in arresting the course of an extension of cholera into the basin of the Mediterranean and into Europe. If the outbreak in Morocco were a derivative from the outbreak in the Hedjaz, the example fails; if it were an independent outbreak, of what advantage is quarantine at Suez or elsewhere at all? Finally, if it were not an outbreak of cholera, we shall clearly have to widen our conceptions of choleraic disease.

ON THE INFLUENCE OF FAIRS, FLOODS, FAMINE, AND SEASON, ON THE DEVELOPMENT AND DISSEMINATION OF CHOLERA.

By Surgeon-General JOHN MURRAY, M.D.

Read: March 2nd, 1881.

In studying the history of cholera in India, and the influence of fairs, famines, floods, and season, on its development and dissemination, it will be best to commence with the consideration of the facts that have been recorded by professional experts; who have had ample experience, whose intelligence is acknowledged, and whose honour is undoubted. From these facts different opinions may be formed by different individuals without altering their value, any more than the absence of other evidence, or negative evidence, can disprove them.

I have obtained the facts referred to in this paper from the records in the Medical Departments of the Presidencies of Bengal, Madras, and Bombay, since 1803; and from the Reports of the Sanitary Commissioners of Bengal, Madras, and Bombay, of the North-West and Central Provinces, the Punjaub, Oudh, and Assam; from many Medical Officers of all the Presidencies, and from my own experience of thirty-eight years, during which I devoted especial attention to cholera.

Under the old organisation of the Indian Medical Service, the Statistical Returns were carefully made out by the Medical Officers, and recorded by the Secretaries of the Medical Board. Since 1871 the reports of the civil population from the various Governments have been carefully compiled by Dr. Bryden, the Statistical Officer, who, since 1864, had shown great zeal and ability, and as a statistician deservedly holds the highest place. His recent death we have all great cause to regret.

The reports of Drs. Cornish and Townsend are extremely important. Their opinions, which are the result of much elaborately conducted observation, are entitled to every respect. To me they are especially valuable as coinciding with

the opinions I have held for many years, and tested during the frequent recurrence of the disease.

It is not my intention now to enter upon many of the most important points regarding the nature of the specific entity or germ of cholera, its action on the human system, or the pathological changes induced by it; nor to dwell on the means of warding off its attacks, or remedying the disease when it appears. I shall limit my observations to the influence of certain customs, and accidents of local floods and famines and seasons in general, in causing the development and dissemination of the disease.

The first subject to which I would draw attention is that of fairs. "Jatra," or assemblages of pilgrims at certain sacred places, have been the custom in India from time immemorial. The medical profession in India has long been alive to the danger of these fairs, and it is pointed out in the first Reports of the Sanitary Commissioners of Bombay and Madras for the year 1866, and the subject is prominently mentioned in the subsequent Reports of the Sanitary Commissioners of the North-West and Central Provinces, the Punjaub, Bengal, and Assam.

The Ganges is held sacred throughout the whole of Hindostan, and pilgrimages are frequent to its source amidst the snows of the Himalayas at Gungootree; to its entrance into the plains at Hurdwar; to its junction with the Jumna at Praag, or Allahabad. These are the most celebrated places of resort; and they are visited at all times, but especially in the early part of the year, which is the most healthy season for travelling.

At certain periods there are jubilees at these places, at which special privileges are conferred. At Hurdwar every twelfth year is considered especially sacred, and the crowd accumulated on the bare ground at the banks of the river (which in ordinary years amounts to from 30,000 to 50,000), will, on these occasions, rise to from one to three millions of people. During one of these jubilees, which are called the Koom ke Mela, in 1783, cholera appeared and killed 20,000 of the pilgrims in one or two days.

In the record that we have of this event it is mentioned that it ceased on their dispersing, and did not spread to the surrounding country. It is also noticed that the village of Juwalpore, which is four miles distant, was not affected. This village still exists, and it equally escaped the attack of 1867, though it was slightly affected in 1879. It is probable that the record of the disease not having spread in 1783, may be owing to the absence, in those days, of statistical reports of the sickness of the civil population.

In the year 1861, there was a similar outbreak of cholera at the Mahadeo Fair, in February, in the Panch Murree Hills in the Central Provinces. Twenty thousand pilgrims are said to have died here in a few days from cholera; but when they dispersed the disease appeared with great intensity within a week in all the surrounding districts. Again, in 1865, in February, it reappeared with the same intensity at the Mahadeo Fair; but the pilgrims dispersed rapidly, and fewer died on the spot. The disease, however, appeared within a week in the surrounding districts, and from thence extended to the whole of the Central Provinces, causing great mortality. On this account, therefore, at the recommendation of the Commissioner, Mr. (now Sir R.) Temple, this fair was interdicted by Government, and the holy well in which the pilgrims used to bathe with such eagerness is now only visited by tigers and hyænas, and from that date no fresh cholera has emanated from it.

During the Koom ke Mela of 1867, at Hurdwar, I was at Meerut, in its vicinity, and, on the cholera occurring amongst the pilgrims, I caused a circular to be sent to all the civil surgeons in the N. W. P. and Punjaub, calling on them for a report of the date of the first case of cholera in their district amongst the pilgrims and residents, with a weekly return of the progress of the disease. By this means its progress could be traced, and an accurate report secured on which reliance could be placed; which would not have been the case had the investigation been instituted after the epidemic had ceased. Some of the civil surgeons received the circular before the disease appeared in their districts. From their reports, and those of the Deputy Inspectors-General and military surgeons, I drew the material facts contained in my report on the Hurdwar epidemic of 1867. The progress of the disease was traced in all directions, radiating from Hurdwar as a centre. It extended for 200 miles in an easterly direction to Oudh; and for 1,000 miles in a westerly direction to Kurachee, and subsequently in a northerly as far as Cabool and Cashmere. The progress of the disease was at the ordinary rate at which the pilgrims travelled along the high roads of communication. Each of the civil surgeons reported that the first case was in a pilgrim, or one who had had intercourse with pilgrims, generally relations and friends, and from these the disease spread over the town, and extended in a few days to the surrounding country. After the subsidence of the attack following the arrival of the pilgrims, there was a subsequent outbreak, or perhaps a recrudescence, on the approach of the rainy season. The total mortality in

the Upper Provinces and Scinde was 73,000. The number of pilgrims was estimated at between 2½ and 3 millions. Extensive sanitary arrangements had been made for their accommodation. There were only a few cases of cholera before the bathing day, the 12th of April, and the general health of the crowd was satisfactory. The weather had been clear and cool till the previous night, when there occurred a severe thunderstorm and fall of rain, with great depression of temperature. This, doubtless, caused much misery to the poor exposed pilgrims, who, next day, started on their homeward journey, liable to be depressed by the reaction following the religious excitement of the ceremony; and many of them scantily supplied with food. I saw them streaming along the road for nearly a week; like a crowded street in London, only all going one way. The road was lined with dead bodies, which were buried by the police, or burned by their relations, or thrown into the canal or river. The ordinary fairs at Hurdwar are attended by a smaller number of pilgrims, from 50,000 to 80,000 perhaps.

There were two slight epidemics in 1872 and 1875, which spread with the pilgrims in a similar course; but over more limited ground, and with less mortality. There was little trace of the disease during the fair, and it did not extend on the east and south-east lines, but spread to the south, *viâ* Kurnaul, as far as Sirsa, and on the west, *viâ* Umballa, to Lahore and Kohat; but not extending to Mooltan or Scinde in either year. In 1875, the attack again avoided the eastern and southern course, and proceeded, *viâ* Saharunpore and Umballa, to the Punjaub, but did not cross the Jhelum, or extend to Mooltan.

The history of the Koom ke Mela of 1879, is a counterpart of that of 1867, the only difference being that there was a slight outbreak in one of the encampments of a hill tribe, on the outskirts of the crowd, four or five days previous to the bathing day, and there was no sudden change of weather, as by the thunderstorm of 1867. The number of pilgrims who attended was less than in 1867, viz., 800,000; and the mortality was also less, viz., 47,000. After the dispersion from Hurdwar the simultaneous appearance of the disease, with the arrival of the pilgrims, is marked in all the districts. In nearly all of them there had been no previous case during the year. In the Punjaub and Scinde there had been no cases during the previous two years.

The date in which the disease appeared in the various districts in 1879 coincides in some to a day with that of the attacks of 1867, and only varies a few days in others; except in Scinde. There is a slight acceleration in most of the Pun-

jaub districts, but in Scinde the acceleration amounts to two months. The course of the disease is identical, but the pace is accelerated. The railway had in the meantime been introduced, and the number of cholera cases, either dead or dying, removed from the trains, amounted to more than 100; and, doubtless, many more who were only in the earlier stage of the disease were transported in the trains, though not noticed by the officials.

Any deviation from the progress of the disease in non-epidemic years must depend upon some exciting cause; and this cause is shown to be manifestly connected with the Hurdwar pilgrims in 1867 and 1879, when the appearance of the disease coincided with the arrival of the pilgrims in each year, with a total absence of the disease up to the date of their advent in that year; and, in most instances, for one or two years previously. I have no record of the year 1866.

Dr. Cunningham, in his report on the epidemic of 1879, says he considers it more probable that the pilgrims were attacked by the aërial poison which was previously located in the village, and affected the pilgrims as predisposed to the disease; so that instead of the pilgrims infecting the place, the place infected them. As in no instance did the supposed localised poison affect a single individual before the arrival of the pilgrims, while many suffered immediately afterwards; I do not say this explanation is not possible; but it is so improbable that few will concur in his views.

The cause of this marked course of this disease in these provinces being so clearly traced, if we find it pursuing a similar course in other provinces it may reasonably be attributed to the same source. Following the course of cholera in Oudh, the eastern part of the N. W. P., and Behar, from the year 1867 to 1879; of these years three were epidemic, viz., 1869, 1872, and 1875, in which, from a quiescent state, the disease suddenly starts into violent action in March.

The great religious fair at Allahabad is held in the end of February, and the time of the appearance of the disease has been recorded in each of the districts within 300 miles of that place. The difference between the recorded facts in this case, and those of the Hurdwar Fair, is that the disease appears six weeks earlier, and the fair is held six weeks earlier. There is no change in the season at this period, nor any other appreciable cause for this sudden development of the disease, and, as we have seen an efficient cause for the Hurdwar epidemic, it may be inferred that a similar cause was in operation in this instance also.

The number of villages in each district, and the number

attacked with cholera in the latest epidemic years have been shown with regard to Hurdwar. The total number of villages was 68,500, of which 8,052, or 10.18 per cent., were attacked. The relative number of persons attacked in each village has also been shown; but I have been unable to obtain this particular, with regard to the other epidemics, as, in compiling the facts from the monthly returns of the Sanitary Commissioners, I found, that when the disease was severe, the same villages were returned for two consecutive months. The result, however, appears to approximate very closely in all India, and this forms an important fact in reference to the nature of cholera.

In the Hurdwar returns there are 68,500 villages, of these 8,052 were affected; and in those affected there was only the proportion of one death to each village in 2,857 villages, two deaths to each in 973 villages; and, taking the whole number, there were not more than two deaths each in 3,800 villages—nearly one-half the number affected. This shows the general diffusion of the disease, and the comparative slightness of the danger of death, and the improbability of these diffuse solitary attacks in the villages having arisen from the general state of the atmosphere. In other instances the virulence of the attack was much greater. In 339 villages there were upwards of twenty deaths; and, in a few individual instances, the villages were decimated and deserted. The causes of this exceptional mortality are, in general, crowding and non-sanitary conditions.

As these religious fairs are evidently one of the most prolific causes of the diffusion of cholera, which produces such great mortality amongst the population, it is desirable to obviate or lessen the danger. There are two parties interested in this question, the pilgrims and the population. Pilgrimages have been resorted to from the earliest times, They are highly estimated by devotees, as affording religious excitement and improving the bodily health by change of air and habits. They are promoted by the priests for religious as well as material purposes. The Hindoo who has visited a sacred shrine in India, the Mussulman who has journeyed to Mecca, have an honorary title amongst their people; they wish them success when they leave on their pilgrimage, and many of their friends would willingly join them if their means allowed. This feeling is of a weighty consideration in a political point of view, and the general adoption of the means employed to ward off the danger by Sir R. Temple at Mahadeo, would lead to great discontent, which must be avoided; and other, though less certain, re-

medies suggested by the advance of medical science should be brought into action. These suggestions point in the first place to sanitary arrangements. It may be said that these arrangements, which have been carried out with great zeal and at considerable expence, have not hitherto been very successful, from the result shown in the two Hurdwar epidemics. Still, in other situations, where the crowd has been less unmanageable, the success has been manifest. The want of success depends more on the execution than the principle. The crowd assembled in 1867 numbered about two-thirds of the population of London; and that in 1879 about one-fourth. When we consider the sums of money that are expended and the sanitary staff employed in London, we may inquire, how is it possible that such efforts can be extemporised on a barren sandy plain, and for a temporary purpose? It is on this line, however, that remedial measures will have to be attempted. They will be expensive, but those who benefit from the pilgrimage would more willingly pay the cost than be deprived of the time-honoured privilege. They undoubtedly think little of the danger to themselves, and if they alone suffered, the case would not be urgent. The danger to the population, which we see exists, from the course of the two Hurdwar epidemics and the great mortality in all parts of India depending on similar causes, proves that the question deserves serious attention.

I shall now proceed briefly to the consideration of the second part of my subject, viz., the influence of floods on cholera epidemics. The facts concerning this part of my subject are based upon the deaths from cholera in Lower Bengal, or the Delta of the Ganges and the Brahmapootra. The facts show the continuation of the epidemic from the end of one year to the beginning of the next. This would be lost in examining the returns of an individual year; when it would appear that there is always a spring cholera, whereas it is merely a continuation of the attack which commenced in November. This attack is peculiar to the Delta of the Ganges—the cradle of cholera. It appears in force at a season when the disease is quiescent in other parts of India, and the cause appears to be clearly indicated by the nature of the country. It is low and on the seaboard; subject to severe storms, or cyclones, in September and October, as well as to the ordinary tides—and it is also submerged by the overflow of the rivers from May to August. The period of the disease here differs from that in other parts of the Bengal Presidency which lie beyond the Delta, as Behar and Orissa and Assam, which approach the dates in the

Upper Provinces. The facts show an increase in the disease in the Delta in March and April, as in other parts of the Presidency; and this increase probably depends on the same cause, viz.,dissemination by the pilgrims from Jugger Nath, which is the most revered shrine on the east coast. Dr. Townsend, the Sanitary Commissioner of the Central Provinces, frequently mentions in his reports that cholera had been imported by pilgrims from Jugger Nath; and it is said that the vicinity of the road most frequented by them is often deserted by its inhabitants when it is reported that the returning pilgrims are suffering from cholera. This spring attack is distinct from the specific attack in the Delta of the Ganges.

The monthly mortality from cholera in the cyclone year 1876-7 was an exaggeration of the ordinary mortality. There was a severe cyclone on the 31st of October which swept a storm-wave of salt water, from ten to twenty feet in height, over the districts of Chittagong, Nowkolly, and Backergunge, 190 miles in length, and extending from 20 to 70 miles in breadth. It submerged the houses, the inhabitants, and the cattle; and immediately subsided, leaving the wrecks of the houses and innumerable dead bodies of men and animals; which, rapidly decomposing, tainted the air with putrid gases. From July to October inclusive, there had been 151 deaths in Chittagong, 119 in Nowkolly, and 573 in Backergunge. Two or three days after the cyclone, cholera broke out with great intensity, and in November there were 2,101 deaths in Chittagong, 4,744 in Nowkolly, and 1,134 in Backergunge. In this and the three following months there were in these three districts 75,450 deaths from cholera. In the four contiguous districts the deaths during the same period amounted to 27,935. There was a similar increase in the other districts, but to a less extent. The returns from the other years show that this is merely an aggravation of the disease, usually found in its cradle; but it clearly indicates the cause of its greater development. There was a partial cyclone with a storm-wave on the southern part of Chittagong in October 1879, after which, in the same manner as in 1876, the disease assumed excessive virulence two or three days after its subsidence. There were 63 deaths in October, 630 in November, and 1,354 in December. In no month of the year is cholera entirely absent from the districts in the Delta. It must be observed that the flood is of a peculiar character, viz., salt water; and that these floods are of frequent occurrence, though in a less degree. Possibly the ordinary tides may

influence the development of the disease. I consider it probable that the seed of the disease is dormant in the soil, and merely called into action in a virulent form by the inundation; not that the salt water originated the disease.

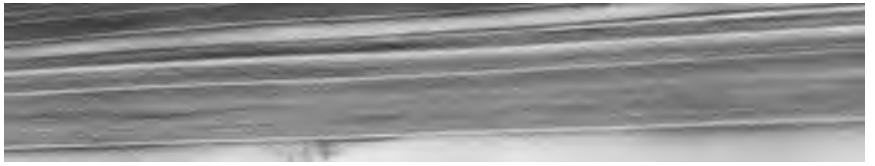
I base my observations on the influence of famine on cholera on the Reports of the Sanitary Commissioner of Madras, by Dr. Cornish, one of the most experienced officers in India. In the year 1866 there was a severe famine in Madras, in which the mortality amounted to 197,977 from cholera; but it gradually subsided in 1867, when the total mortality was 32,591. In the years 1873-4 there was scarcely any cholera, the total number of deaths for the two years amounting to 1,153 only; and this exemption continued till March 1875, when severe famine commenced, which lasted during the two following years with a total mortality from cholera of 617,879. The absence of the disease during the two preceding years indicates that a new cause had come into action; and, as it was reported in 1866, and also during the later famine of 1875-6-7, that the mortality was greater in those districts where the price of food was highest, the connection between the famine and the mortality appears indisputable. It is the opinion of the Sanitary Commissioner, in which I fully concur. The famine extended to Bombay with a similar result. The facts relating to the Madras and Bombay Presidencies clearly show that the conditions under which famine occurs help to predispose the people to fatal attacks of cholera; and we know that the conditions of bad and insufficient food, scant and doubtful water-supply, overcrowding on public works and resting-places, and concentration of the poverty-stricken over a limited area, under any circumstances, conduce to the disease; and, in addition to these concomitants of famine, the transmission of the disease by human intercourse, would add to the general diffusion, and account for its great fatality.

The influence of season on the development of cholera has been treated by me in a former paper, and I shall now only refer to the facts relating to the subject. These extend over a very long period, but include a much smaller number of people, and these under very different circumstances as regards sanitary arrangements and medical treatment, which must materially modify the course of the disease. The facts include the habits of the disease among European and native troops and native prisoners. They are not subject to two of the influences that have been described, viz., famine and flood; but season applies generally to all, and the modifications from the quiescent state must be influenced by it.

tlidge, 200 miles round Hurdwar,
d 1879.

PUNJAB AND SCINDE.

					1879.				
Villages affected.	First Case of Epidemic.	Duration of Epidemic.	Total Deaths.	Ratio of Deaths in Villages affected.	Villages affected.	First Case of Epidemic.	Duration of Epidemic.	Total Deaths.	Ratio of Deaths in Villages affected.
108	April 20	7	647		19	April 14	5	87	4.6
159	„ 19	7	957		25	„ 19	5	82	3.3
352	„ 24	5	2,213		309	„ 21	4	1,947	6.3
584	„ 21	7	3,419		71	„ 15	7	868	12.2
48	May 1	8	284		14	„ 19	2	20	1.4
349	April 21	7	2,095		269	„ 15	7	1,673	5.8
111	„ 20	7	664		12	„ 21	3	42	4.0
295	„ 20	7	1,773		147	„ 21	7	2,048	13.9
283	„ 28	5	1,696		66	„ 18	6	430	6.5
64	„ 30	3	383		59	„ 20	5	380	6.4
14	„ 30	6	850		293	„ 20	7	1,962	6.7
...	...	...	...		53	May 20	5	176	5.4
315	May 7	5	1,892		601	April 20	7	2,914	4.9
179	August	5	1,072		12	Aug. 9	3	39	3.3
358	April 25	6	2,147		73	April 24	4	346	5.0
205	July 26	3	1,229		107	May 18	5	692	6.5
582	May 11	6	3,493		138	„ 1	6	1,002	7.3
105	July	6	632		22	June 17	4	164	7.5
576	August	4	3,467		227	May 21	7	1,359	6.0
33	„	3	196		...	„ 24	3	99	12.4
23	„	2	135		220	„ 19	3	1,433	6.6
...		...	...		13	„ 22	2	74	5.6
93	Aug. 1	6	557		176	„ 9	4	1,174	6.7
4,706			29,906	8.0	3,317			20,096	6.0



SUMMARY.

In Scinde there have been three severe epidemics since 1866, whilst the intermediate years have been entirely free from cholera. Two of these epidemics, those of 1867 and 1879, have been shown to have depended upon pilgrims from the Koom ke Mela at Hurdwar, on the Ganges; the third, in 1869, was connected in the same way with the pilgrims from the Koom ke Mela at Toura, on the River Nerbudda, in Broach. This jubilee fair takes place at the end of April, once in nineteen years; and 1869 was a jubilee year, and 400,000 pilgrims attended the fair. There had been a few cases of cholera in this district since the beginning of the year, and in April there were 84 cases, of which 35 occurred at the fair. Cholera had been very prevalent in the neighbouring district of Surat; but in the other surrounding districts there was very little cholera till after the dispersion of the pilgrims at the end of April. In May, cholera broke out with great intensity in the five districts situated within a hundred miles of Broach, and extended in a northerly direction towards Scinde, reaching Thur Parkur, 230 miles distant, in June; Hyderabad, 390 miles, in August; and Kurachee, 550 miles, in September. The average rate at which cholera progressed from Toura to Kurachee in 1869 was six miles *per diem;* the rate from Hurdwar to Kurachee in 1867 was nine miles, and in 1879 thirty-one miles *per diem;* the acceleration of speed in the last instance being attributable to the introduction of railway travelling.

In the six severe epidemics that have appeared since 1867 in the twenty-three districts surrounding Hurdwar, extending to a radius of 200 miles, 61.28 per cent. have commenced in April and May, whilst in twenty-five districts covering a similar area round Allahabad, 65.55 per cent. commenced in February and March. During these months, only one epidemic commenced in the Hurdwar district, though these districts are contiguous. It must be remembered that the sacred day at Allahabad occurs in the end of February, whilst that at Hurdwar is in the middle of April. In the districts in the vicinity of Hurdwar there were 17.73 per cent. of attacks in June and July; whilst in the months of April and May there were 32.22 in the Allahabad circle. This increase in the ratio of attacks in the latter is probably owing to the assembling of pilgrims at a very sacred shrine, Adjoodai, in the middle of April, or at the holy city of Cassee, as the natives call Benares.

NOTE.—The outbreak of cholera in Mecca in November 1881 bears a strong resemblance to the outbreaks at Hurdwar. The disease commenced with great intensity the day after the great sacrifice of sheep at Mount Ararat on the 2nd of November, and it appeared within a few days in the surrounding cities of Medina, Jedda, etc. There are reports of its having appeared at Elwedj. I anticipate that it will continue to follow the course of the pilgrims in Africa, and to Damascus and Constantinople.

These returns are little affected by the pilgrims who exercise so strong an influence over the general population. Season will not produce the disease any more than famine or flood, but it predisposes to an epidemic, and brings into force the ordinary agencies, which we have seen to disseminate and predispose to the disease.

The mortality from cholera varies in the different Presidencies; it is most sharply defined where the seasons are most distinctly marked. The three different classes of military and civil who are under medical treatment are simultaneously affected, but at a different season from the civil population, who are subject to the influx of the Gangeatic pilgrims.

CERTAIN CONSIDERATIONS REGARDING CHOLERA AND FEVER.

By Surgeon-General C. A. GORDON, M.D., C.B., Honorary Physician to the Queen.

[*Read*:—*February* 2, 1881.]

Mr. President,—1. *Health Conditions in India.*—Here is an exact transcript of a weekly Health Report* of India, recently given in one of the public papers devoted more especially to information regarding Britain's greatest dependency, namely:—"*Bombay:* Health good. *Bengal:* Fever prevalent in twenty-four Pergunnahs, Moorshedabad, and Rajshaye, Burdwan, Rungpore, and Purneah; cholera still in Cuttack. *North-West Provinces and Oude:* Health good, except in Bareilly, where fever and cholera (thirty deaths) have appeared. *Punjaub:* Health fair. *Central Provinces:* Fever still prevalent. *British Burmah:* Rangoon and Akyab, health good; Bassein, a little small-pox; Prome, cholera decreasing. *Assam:* Health fair. *Mysore and Coorg:* Fever prevalent. *Central Indian States:* Morar, cholera disappearing; Rajpootana, Mewar, fever prevalent. *Others:* good."

2. *Examination of Particulars.*—Let us examine the particulars just given, and these are the results which present themselves. The general health condition throughout India was unusually favourable; with the exception of places named it was good; at places far asunder as the Punjaub and Assam, "fair", that is, not absolutely good, and yet disease not prevailing to an extent to attract special attention. Elsewhere, however, one or other of the three forms of disease which most prevail throughout that country existed at different places, although, fortunately, with inconsiderable intensity, as compared with their manifestations in the same localities on many previous occasions. Thus, fever was prevailing throughout the stations of Lower Bengal; also far away in one direction in Mysore and Coorg, and far away in another in Rajpootana; it lingered in the central provinces, while at Bareilly, far away in a third direction, fever and cholera had both appeared, the advent of the latter malady

* Week ending 8th October 1880.

signalled by the death of thirty victims. Elsewhere cholera alone is reported as present; in Cuttack as still prevailing; at Prome as decreasing; at Morar as disappearing; while, at an extreme corner of the Empire, near one of the estuary mouths of the Irawaddy, the record says there is a little small-pox at Bassein.

3. *Deductions.*—That is, during the same week health-conditions at the several parts of our Indian Empire differ absolutely from each other—a circumstance in accord with existing differences in physical condition and aspect of places far apart from each other; differences in climate and seasonal changes; differences in kind and condition of vegetation at the same time in those places; differences in the conditions of their several populations; and, doubtless, also to the operation of certain great ulterior influences as yet only cognisable in their effects, but not solvable by the theory alone that a specific organic poison introduced from somewhere else, or suddenly vegetating on the spot has, for a time, exerted its evil and deadly power upon humanity, then, itself, becoming dormant, asleep, disappearing or passing onward in its work of devastation—the manner of its disappearance or progress as unaccountable, according to the same theory, as were the circumstances of its original development.

Two months elapse. At the end of that time the health-conditions are thus reported:*—*Bombay:* Fever continues, but is abating in Guzerat. *Bengal:* Fever still very prevalent in many places, and deaths reported very numerous in Nuddeah and Jessore. Cholera in Cuttack, Pooree, and Maldah, and some cases reported from Budruck and Deoghur. Small-pox in Pooree, Chumparun, and at Jhaldah in Manbhoom. *North-Western Provinces and Oude:* General health good, with the exception of slight fever in Bareilly and Agra, and some cholera in Fyzabad. *Central Provinces:* Good. *British Burmah:* Cholera in parts of Pegu, but few fatal cases; a little small-pox, otherwise public health good. *Assam:* General health fair. Fever about Gowhatti. *Mysore and Coorg:* Health good.

We compare the general results of these two health reports thus:—At Bombay, where health was good at the date first referred to, fever has, in the interim, occurred, and continued to prevail. In Bengal, both cholera and fever still prevailed in the localities formerly named, with two exceptions, viz., Nuddeah and Jessore. Cholera had ceased in Cuttack, but appeared in Maldah, Budruck, and Deoghur. Small-pox oc-

* For week ending 9th December 1880. See Allan's *Overland Mail*, 29th December 1880.

curred in several places previously free from the epidemic, particularly Pooree, Chumparun, and Manbhoom. In the North-West Provinces and Oude health continued good as before. At Bareilly, where fever and cholera previously prevailed, only slight fever was reported; cholera had left that town, but appeared at Fyzabad. In the Central Provinces, where fever formerly prevailed, the population had become healthy. In Burmah the state of health was unaltered; but in Mysore and Coorg, where fever had prevailed, the state of health had become good. Thus, in what may be termed the ebb and flow of these three diseases, indicated by the reports before us, we find ourselves face to face with some of the great problems of epidemiology.

4. *Method of Inquiry.*—If, then, we endeavour to follow the natural history of these scourges of mankind, either on the occasions now referred to, or on the others, when, with varying degrees of intensity, they have swept over continents, it seems to me that such endeavour is more likely to obtain that measure of success incidental to the pursuit of subjects from their very nature abstract and abstruse, if we take a comprehensive view of the various conditions under which they severally or together occur, than if we confine attention to one set of those conditions to the relative omission of others perhaps less tangible. If organic pollution alone of air, earth, or water be the cause of cholera, how are the sudden appearance, culmination, decay, disappearance, absence for long periods of these diseases, to be accounted for, conditions of air, earth, and water meantime remaining unchanged? How similarly with regard to fever, or a particular kind of fever? How far the circumstance that fever and cholera on one occasion prevail independently of each other; on another contemporaneously; on a third alternating with each other; individual cases of fever passing into cholera; others, at their onset presenting the ordinary symptoms of cholera, developing into fever; and yet a third set, at their commencement, manifesting symptoms characteristic in equal proportions of both those forms of disease? How, with regard to an acknowledged infectious and contagious disease, viz., small-pox, on the first occasion named, limited to a spot of inconsiderable size, remotely situated in the Eastern division of our Indian Empire; on the second disappearing there, but occurring at places completely apart from each other, as are Pegu, Pooree, and Chumparun, to the exclusion of the vast intervening territory between those three localities? By what explanation do we account for its being so limited in both these instances to the localities named; whereas, on other

occasions, it spreads onwards directly from place to place, within certain limits, devastating populations in its course? Are these several phenomena to be accounted for simply by the theories of organic pollution and contagion? For my own part, I say—No; they are not. And, from this point of view, I proceed with my further remarks.

5. *General Considerations.*—The teachings of past experience point to the circumstance that the law or laws, in accordance with which small-pox has its periods of prevalence and of dormancy, are not much dissimilar, if at all, from those by which cholera is regulated. We have been informed that particular forms of disease comprised in what may be termed the small-pox group, have their corresponding rise and fall. It is not assumed that actual affinity exists between that disease and cholera. On the contrary, there is this great difference between them—that by means of vaccination* individual susceptibility to small-pox becomes limited; that to cholera is decreased by any similar or allied process. But, as regards cholera, so with small-pox, the bulk of evidence is to the effect that, so long as the general epidemic of either disease is active, and what, for want of a more definite name, is indicated as diffusive energy remains in full operation, isolation of individual cases does not retard, far less prevent the expansion of morbific influence over the population of what French authors appropriately describe as the epidemised locality. Neither has it in bygone times been found practicable, even by means of inoculation, to extend small-pox during the intervals between periods of epidemicity.†

More intimate is the relationship which phenomena of cases and manner of prevalence of both cholera and fever, as observed more especially in India, justify the belief that there exists between these two diseases. On this subject one of the very recent writers‡ on Indian diseases expresses himself in this way:—"Owing to the marked association of epidemics of cholera and fever in Northern India, and to the

* Is it the case, as stated by Copland, that, as small-pox has decreased since the introduction of vaccination, scarlatina, measles, pulmonic, and cerebral inflammations have increased?—Art.: "Epidemics"—*Dictionary*.

† As a foot-note I mention that in India the deity of cholera is Maree, or, as the name is respectfully alluded to, Maree-Ama; her handmaid is Seetala, goddess of small-pox. In China the deity of small-pox appears to be Niang-Niang; she also must be mentioned respectfully. Thus, when in 1875, the late Emperor died of it, the *Pekin Gazette* announced the event as "the enjoyment of the heavenly flowers". The Chinese theory of small-pox and measles is that they depend upon a poison inherited from the parents, which resides in the system till excited by external causes, like fire concealed in the flint.—*Customs Reports*, January —March 1875, p. 35.

‡ Macnamara, p. 120.

frequent similarity, especially on the North-West frontier, of bad cases of malarious fever to cholera seizures, some observers have been led to ascribe the two diseases to an identical cause." He adds—"The identity of the miasms of cholera and fever is not pleaded, but only their similarity, which is chiefly shown in the external conditions that favour them both." Northern India* affords many examples of the impossibility of predicting from the physical conditions of a locality the degree to which fever will flourish there.† Elsewhere throughout India a similar observation is applicable with regard to cholera, and the circumstance is recognised, that in localities noted for the occurrence of malarial fever, diarrhœa, dysentery, and affections of the abdominal organs generally prevail. Even in England, cholera, when it has occurred, has been most severe in low-lying places, such as were formerly the favourite homes of ague, and such as have subsequently furnished numerous cases of what has been described as "paludal enteric fever", a mixed type of malarial intermittent and typhoid;‡ in other words, malarial fever, attended by intestinal complication. In India the occurrence of bowel affections, with and without other organic lesion, and with and without fever, has as distinct an apparent relation to season, as is presented by the course of vegetation in the same locality, or, indeed, any other natural phenomenon. So, also, in respect of fever distinctly "ardent" in type attended by cerebral complication; so with that, the complication of which is pulmonic. Fever, diarrhœa, and dysentery§ in that great country prevail simultaneously or alternate with each other. In all, affections of the spleen, the liver, as also of other glandular and glanduloid tissues, sooner or later, occur as complications. In many instances ostensible causes of illness are apparent, but in many others, such causes as are usually assigned fail altogether to acount for their occurrence.

* Dr. Livingstone relates that in some parts of Central Africa he found some dry, sandy, and clean tracts, more dangerous, as regards fever, to encamp in than others covered with reeds and rushes. Mr. Hyde Clarke mentioned an instance of a town in Africa built in an healthy situation, and healthy until cultivation began. Three years afterwards fever broke out, and ran through it all. † Macnamara, p. 121.

‡ Russell, p. 28.

§ Sir Joseph Fayrer, in his Lettsomian Lectures, 1881, mentions that in India dysentery is often accompanied by malarial fever; that among the complications of the former is the typhoid form; that a close analogy exists between dysentery, diphtheria, and typhoid fever; that the disorganisation, although principally in the large bowel, has a tendency to pass the ileo-cælic valve.—*British Medical Journal*, 29th January 1881, p. 149.

Types and Unity of Fevers and Cholera.—Both cholera and fever take place under various circumstances and conditions —individual and general. They also present, in particular instances, various types or modifications in their own special characters. Thus, cholera is met with in one set of localities in India, occurring in isolated cases throughout all seasons, those isolated cases in certain seasons being the precursors of a general outbreak, in others not so; at times apparently induced by unwholesome articles of food or drink, at others following the administration of therapeutic evacuants. In a second set of localities cholera occurs in the shape of regular outbreaks at certain intervals of time during the intervening periods, all general conditions remaining precisely as they are during those outbreaks, and even sporadic cases so extremely rare as to be considered almost absent. When outbreaks of cholera do occur, they present certain modifications in their phenomena, these modifications observable when the epidemic of one year is compared with those of preceding years, the characters of individual cases with those of others; and even in certain instances where direct importation of the disease has been traced, assuming new, and it may be exaggerated, characters, in its new locality. Then, again, the disease presents various characters in individual cases—congestive or algide in one, asphyxiated or spasmodic in others; rapidly destroying life in one set, more slowly in another; the attack sudden and without premonitory warning in one; in another, preceded by malaise or diarrhœa, or both; sometimes for a few hours, at others for some days. But neither in designating these several modifications, or others here unenumerated, in which cholera occurs, do we find a term applied in a specific sense, either as implying a particular manner of causation, a particular series of pathological changes —although such do occur—or a particular method of treatment to be followed. In all these fevers cholera is simply recognised under that general term, causation being looked to in reference to public measures of mitigation, and with a view, whether to be realised or not, of future prevention of a similar outbreak.

With regard to fever,* so various are the characters and modifications, according to geographical position, climatic

* Dr. Chevers records the occurrence in India of Pali plague, and relapsing fever, in 1815-19; of cholera in 1817. He states, in reference to the severe epidemic of fever which prevailed in the gaols of Agra, Umballah, Futtyghur, Allyghur, Jeypore, Malwa, etc., in 1864, that it was variously described as true typhus, contagious fever, typhoid fever, and relapsing fever, in the same year of the Burdwan fever epidemic.

conditions, seasons, etc., acting upon individual diatheses, temperaments, habits, and other personal circumstances, that while, as already related, ancient Hindoo physicians reckoned thirteen different kinds, this category has in more recent years, on the one hand, been so expanded as to embrace some three hundred different species; on the other, it has been so compressed as that only one fever is acknowledged to affect humanity, this one fever presenting so many modifications according to circumstances and conditions as related, yet itself preserving its unity in this multitude;—fever, with this modification here, that other there, and so on.

Partly as a result of improved sanitation, partly also of a circumstance which has more than neutralised all sanitary measures that have been adopted in India since 1864, attacks of fever among our troops in that country are, in the mass, less violent, less acute, less ardent than in days before the great Sepoy Mutiny. On the one hand, measures for preserving health, begun by army medical officers, while sanitary science was yet recognised only as "medical police", have been developed upon the lines then laid down; space in barracks has been increased three-fold, intemperance diminished, inducements created for men to remain in or near the barracks, instead of wandering. But against all these, and outbalancing them, is the one measure of short service, with the constant stream into India of young immature lads incidental to it.*

Individual cases from time to time occur in which the symptoms, beginning with hyper-pyrexia, gradually merge into such as are adynamic. In former years, cases of this nature would have been described as of synocha; more recently, but yet a good many years ago, the expression typhoid would have been applied to the latter condition, not, however, in a specific sense, but adjectively; the counterpart, as it were, of ardent, acute, inflammatory, and so on. Still more recently this term, instead of being applied thus adjectively, as expressive of a condition, appears to have a specific significance attached to it, and the resulting turmoil has been considerable. It were well, I think, to indicate

* Article in *Encycl. Britan.* By Professor Traill, Edinburgh. Surgeon-Major Don, in his paper read before this Society (pp. 285 and 286 of *Proceedings*, 1880), observes, with regard to Bermuda, that "the robust, big-chested and muscular man, often a hard drinker, did get fever, but not enteric. The man of altogether reverse physical type and development, often temperate, or even teetotal, was the victim of enteric in an especial degree." Also "that the age at which there is the greatest proclivity to the development of struma, tubercle, and such-like diseases of low nutrition, is that in which there is the greatest liability to enteric fever."

clearly in future Reports in which of these senses, or in what other, the expression typhoid, when applied to a case of fever, is intended to be understood. In some instances, the forms of fever* met with in India admit of being referred to one or other of the headings in accordance with the official "Nomenclature of Diseases". In the great majority this is only approximately practicable. In China, "mixed fevers" are described as such in published reports. In India, cases of fever assigned to climatic causes were, and still are, indicated as "malarial"; this in brief. They are of all degrees of severity; those of greatest violence tending rapidly to arachnoidal effusion and heat-apoplexy—such are, in India, termed heat fever; in China, pernicious fever;† others, as already remarked, passing into cholera. But, in reference to a particular type and complication occurring in India, the question is asked by our highest sanitary tribunal,‡ "Is there such a specific fever as enteric fever at all?"

7. *Specific Origin Theory.*—That certain diseases are due to the action of specific poisons upon the system is a circumstance familiar to, and recognised by, all observers. Syphilis and rabies furnish examples of this class. In both, the poison appears to increase in, and pervade the system, but is itself only transmissible, at least in its acute state, by means of inoculation. Other specific poisons multiply and increase similarly, and are transmissible both by inoculation and infection, including contagion. Of this class are small-pox, scarlatina, puerperal fever, etc.§ That these diseases occur, however, under conditions where neither inoculation nor infection can be traced, or with good reason assumed to have been in operation, is a point forcibly brought to attention by the records of epidemics contained in current medical literature.‖ Certain of these poisons are said to be capable of producing now one disease, now another, accord-

* For views of the older medical officers, regarding fever generally, see my special reports, I, p. 164; and II, 56-76.

† *Chinese Customs' Reports*, July—September 1872, pp. 94-95.

‡ The Army Sanitary Commission. In the *Chinese Customs' Reports* July—September 1873, p. 56, these remarks occur in reference to dysentery and typhoid fever: "Whether the former disease is always specific is fairly open to doubt, and the specificity of the latter is denied by one of the greatest living authorities on the subject."

§ Puerperal fever is one of several diseases which arise spontaneously when puerperal women, wounded men, etc., are crowded in hospitals. See Copland's *Dictionary*, Art. Epidemics.

‖ See *Transactions of Epid. Society*, vol. iv, part iii. Among others, Dr. Longstaff (p. 421) indicates that particular groups of disease prevail, some seasonally, others not apparently affected by season. The diarrhœal group has the greatest mortality in the summer quarter, and is most fatal in years with dry hot summers.

ing to individual conditions and local circumstances, insanitary and otherwise; and, as a result of recorded experiments, certain animal fluids, originally innocuous, have, by artificial means, had their characters so transformed as to acquire such properties and their infective principle, then called pyrogenic. But, according to my view, analogy justifies the belief that, if by artificial means, transformation or evolution of this nature can be induced, equally may it be so by means of the inherent vital forces which regulate and determine the general organism of which organic matter is a part.

By analogy, also, the same or a similar principle applies with regard to various changes which occur in some instances during life, in others after death, and are capable of inducing disease in persons into whose systems matters so altered are introduced. Instances in illustration present themselves in peritonitis, erysipelas, pyæmia, etc.;* in cadaverous poisoning, in the several affections induced by fish and flesh meat in various stages or states of decomposition. But, on the other hand, we trace many illustrative exceptions. Thus, the flesh of animals dead by diseases the most horrible has been eaten—glanders, for example—in a cooked condition, without evil effects; animal poisons have been swallowed without ill effect; black vomit in cases of yellow fever has been swallowed; cholera matters have been tasted, and even inoculated into animals and people, with similar negative results. Hence, I venture to remark, the necessity for caution before we assume that, because a particular circumstance happens at one particular time and occasion, therefore precisely the same circumstance must of necessity occur under all and every combination of conditions. Such an assumption is obviously in disaccord with actualities.

During more than forty years has search been pursued zealously, but vainly, for specific poison of fever or cholera germs. Microphytes, bacteria, vibriones, etc., have each been indicated as the actual cause, each in turn, as a result of further observation, relieved from such onus. Thus, we can only say that cholera as a phenomenon from time to

* Dr. Alfred Carpenter believes that the poison of typhoid fever may arise *sua sponte*; Dr. King that it may be produced *de novo* by the putrefaction of albuminous stools; Dr. Lowe that it may arise from a process of "evolution" in decomposing matters; that inasmuch as puerperal septicæmia, pyæmia, erysipelas, or hospital gangrene may develop autogenically under a given combination of circumstances, and then spread by contagion—why not enteric fever? Dr. Cayley, on the other hand, says the weight of evidence is against the *de novo* theory. It has been remarked that "at bottom we are all humoralists and believe in infection; it is not until we have to say where and how, that questions arise."

time recurs; that it affects individuals and communities; but, in our present state of knowledge, or rather ignorance, of its laws, all circumstances of a positive nature with regard to its prevalence that have been adduced, have their counterparts negatively; all of a negative character, their counterparts positively.

Certain forms of fever are assigned to causation of a specific kind. Those to which attention is now necessarily restricted are such as by custom are referred to "malaria", and such as are referred to a specific poison originating in, and propagated by, means of decomposing animal, more especially fæcal, matters. That certain organisms are said to have been detected in the atmosphere of "malarious" places, may or may not be a fact capable of standing the test of further minute investigation.* Even in the event of their being so, the presence of such organisms in those localities is itself due to the sum of conditions which induce particular and distinctive phenomena of life in the animal and vegetable kingdoms generally, in those localities, not as apparently is implied, themselves the cause of those phenomena. Their own development, judging from published reports on the subject,

* "MALARIA.—At the meeting of the Royal Academy, a memorial was presented, entitled 'New Studies on the Nature of Malaria', by Signors Cuboni and Marchiafava. These studies corroborate at every point the conclusions drawn by Professors Klebs and Tommasi-Crudeli as to the vegetable parasite which produces the malaria fever. These observers have also discovered new facts which may serve to clear up one of the most interesting pathological facts, that is, the contagion of infectious diseases. They have been able to communicate the true malaria fever to animals (dogs and rabbits) simply by inoculating them with blood taken from malaria patients, which means that they have rendered contagious a malady which, under natural conditions, can neither be transmitted from man to man, nor from man to animal. They have also found that the presence of the *bacillus malariae*, in its sporadic form, in the blood of malaria patients, is a constant fact. This parasite is found in great quantities in the blood during the cold stage of the fever, while in the warm stage it has almost totally disappeared, leaving only traces of its spores. These, in their turn, can produce a second generation of parasites. In an appendix to the memorial, the authors quote a letter from Dr. Lanzi of Rome, who declares that he constantly noticed the same fact in the blood of twelve patients in the hospital of San Giovanni in Laterano, examined by himself during the cold stages of the fever.—There was further presented for insertion in the acts of the Academy a communication by Signor Tommasi-Crudeli. In it the author shows the utility of making experiments on the possibility of rendering the human organism impervious to malaria by small doses of arsenic taken daily. He says he has good hopes that he is well on the road to certainty, and describes the methods he has adopted; but, having instituted a new series of experiments on animals without having been able to obtain a final result, he invites all students to undertake similar experiments, and thus contribute to the solution of so vital a problem." That is to say, a theory is, in the first instance, elaborated, in the second, facts in support of it have to be sought for.

is not constant; it depends itself upon conditions which only occur at intervals, and on each occasion are temporary in their duration. Certain organisms, bacteria, for example, differ even according to the age of materials from which they are produced; sterilised solutions were obtained from new hay; non-sterilised, from old hay*—according, so runs the explanation, as to whether they have lost their inherent generative energy.† Another abstract expression, you will observe.

As to the general disease, fever, I take the definition given recently by one of the highest of living authorities. "It is not only a state but a process; the state of fever once established, it may vary in its course, duration, and local inflammations which accompany it indefinitely."‡ According to views now generally accepted, this state or process may be induced by various causes, some of a nature extraneous to the individual, others inherent in himself.

As far as documents at my disposal authorise a conclusion on the subject, the conclusion at which I thus arrive is, that with regard to fevers in India, ardent in their characters, attended by cerebral, pulmonary, hepatic, or other complications, except ulceration, involving the glands of the lower part of the small bowel, observers have seen no necessity to assume the operation of a poison, specific in its nature. But when the latter complication does occur in the course of an attack of fever, with or without the presence of low adynamic, that is, typhus or typhoid condition of the patient, the tendency for some years back has been too much to assume the operation of a specific poison, the actual existence of which remains undemonstrated; too little to consider the operation of general conditions as they affect man, individually or in the mass.

The theory of specific poison is thus expressed by its advocates: "enteric fever arises from a specific poison contained in, and transmitted by means of, sewer emanations"; also, "the poison, the manifestations of which present that group of symptoms called enteric fever, and which, in fatal cases, is invariably found associated with lesions in Peyer's patches and mesenteric glands, is as distinct and separate as any of the other animal poisons which produce specific results, such as small-pox, measles, scarlet fever, cholera". But sewer emanations do not always induce such effects in indi-

* Professor Tyndall, *Proceedings of Royal Society*, No. 181, May 1877, p. 230.

† *Op. cit.*, p. 232.

‡ Dr. Burdon Sanderson, *Appendix to Sixth Report*, new series, by Medical Officer to Privy Council, p. 9.

viduals most constantly and directly exposed to them; precisely similar effects also result in cases of fever due to causes other than these. How, then, can specific results arise from different series of causes, the causes themselves non-specific?

Inoculation of rabbits with blood from typhoid fever patients has produced in these animals not specific typhoid fever, but typhoid septicæmia, precisely as results from inoculation with organic matter in a state of putrefaction.* In this respect there is reason to believe that the Lepuridæ manifest a greater degree of susceptibility than occurs in man. Unfortunate rodents have been inoculated with hydrophobic saliva from the human subject; death has rapidly followed, but, whether or not by hydrophobia, was a point on which the three operators had as many opinions among themselves.† So much with regard to a known specific poison.

As further adverse to this theory of specific poison, time

* M. Decroix, *Hygiene and Surgery of Franco-German War*, p. 226.

† Dr. Davaine (*Courier Méd.*) remarks that, according to the researches of M. Pasteur, we know that putrefaction is the fermentation produced by infusorial animals of the family of Bacteria or Vibrio. The presence of these infusoria is one of the signs which characterise this phenomenon, which, according to Dr. Davaine, may take place as well in a living animal as in the laboratory. But what are putrid diseases? It is in order to reply to this question that the eminent experimenter has made some new experiments, of which the following are a short *résumé*:—

Blood from several patients attacked with gangrene was injected into rabbits without any result. This blood contained neither bacteria nor vibrios. "But if the gangrene", says Dr. Davaine, "be not itself a septicæmic disease, it may become so by putrefaction of the gangrenous parts, and the introduction into the blood of the putrid elements." This is what took place in an experiment of Dr. Bouley, of which we have spoken.

Dr. Davaine has also made a particular study of typhoid fever. In all the cases the blood of a typhoid patient, inoculated on rabbits, has caused death in a shorter or longer time, sometimes in a few hours, sometimes after two or more weeks, and he never injected more than the thousandth part of a drop, so that the septic nature of typhoid fever cannot be doubted. "I would add", said Dr. Davaine, in closing his communication, "that in successive generations of poison obtained from the inoculation of rabbits, I have not met with any difference between typhoid septicæmia, and that produced by the inoculation of organic matter in a state of putrefaction, either in the phenomena, or progress, or termination of the disease. It is to be noticed, however, that the incubation is generally much longer in the first case than in the second; but in another communication he will show that there is nothing in this special to typhoid fever.

M. Pasteur inoculated two rabbits with saliva from a child just dead from hydrophobia, with the result that both these animals died in less than thirty-six hours afterwards. Their blood, after death, when examined microscopically, presented minute organisms, having the appearance of the figure 8; these occurring in masses. These organisms, when intro-

alone restricts the number of my references to recognised authorities on the subject; the names of those authorities appear in my printed reports and other documents. Thus, the *de novo* origin of fever—in other words, the evolution—finds support among the group of observers, names of four among whom are given in my first special report. One writer observes that "fever cannot be traced to any specific poison"; another that, "in ninety-nine cases out of every hundred, the disease is not from any specific germ"; another, that "all medical men are not agreed as to the cause of enteric fever"; another, that "causation is so little understood as to give us little help in the diagnosis of this disease"; several others, that "enteric fever may arise from fatigue and exposure"; a still more recent writer that, "under special and peculiar conditions, enteric fever may become instituted in the system without any external agencies other than such common causes as give rise to that lowered tone of vitality* which favours the springing up within itself of the morbid phenomena in question"; yet another, that "specific causes are not in all cases required to produce an attack of fever—that changes may take place spontaneously in one or more of the functions, and proceed to give rise to the worst forms of fever"; and so on. In India, of the cases investigated by me, the reports by medical officers concerned indicate that in no instance among them was an attack of fever actually traced to the operation of a specific poison. Hence, as a general result of what has thus been adduced, I submit that I have good grounds for the opinion arrived at and expressed by me, that not only is the presence of a specific poison undemonstrated in fevers met with in that country, attended by intestinal complications and characterised by a low, typhoid, or adynamic condition of body, but that such cases of fever occur as the results of general conditions, climatic,

duced into a *bouillon* of veal propagated it rapidly, and when inoculated in other rabbits, induced death similarly as in those from which they had first been taken. But, it is asked, was the disease in either case *la rage*? M. Raynard says Yes. MM. Colin and Dujardin-Beaumetz say No. M. Pasteur says neither Yes nor No.—*Gazette Médicale de Paris*, 22nd January 1881.

* For example, of 23,000 French prisoners confined at Stettin in 1870-71, 93 were attacked with typhoid fever. The outbreak on that occasion was attributed to the combined effects of hardships, defeat, and imprisonment, that is, to the ordinary causes of typhus in armies under circumstances more or less similar.

Dr. Lowe records the case of a man, dirty in habits, and subject to diarrhœa, becoming ill of typhoid fever, in the absence of possible communication with one affected, with water pure, no drains, but his cottage damp, dirty, and overcrowded.

endemic, epidemic, and others, acting upon particular conditions and diathesis of individuals.

This view, I submit, finds support, by analogy, in recent published reports of diphtheria and scarlatina in this country.*

Certain diseases, which undoubtedly in the first instance have arisen without the intervention of a specific poison or poisons, have subsequently spread by means of specific contagion. Typhus fever in camps, dysentery in fleets, ophthalmia in barracks, pyæmia in war hospitals, are examples in point.† But sudden outbreaks at places far apart, of diseases such as we are now considering, seem to me to indicate that they also, although capable of becoming epidemic, and in particular instances of being propagated by contagion, yet, as in the instances before us, their first development occurs autocthonously and autogeneously. In India, cases of fever occur that no theory of specific contagion can explain.‡ The doctrine of specific poison is altogether inapplicable to the history of what is called enteric fever in that country.§ So say high authorities on these subjects.

* See *Brit. Med. Journal*, 18th December 1880, p. 989:—

"DIPHTHERIA AT THE CHILDREN'S HOSPITAL.—A very remarkable, though unfortunately completely negative, report has been made by Mr. W. H. Power, into the circumstances attending the outbreak of diphtheria in the Great Ormond Street Hospital for Sick Children in March of this year. The outbreak in question was without parallel in the history of the hospital. . . . . In-patients, of four out of the five general wards of the hospital, were almost simultaneously attacked by diphtheria, or scarlatina, and this under circumstances that seemed to exclude, at least in the majority of cases, antecedent human infection. Mr. Power's inquiry, though thorough and searching, has not succeeded in demonstrating a cause of the diphtheria. . . . . The five initiatory cases of throat-illness in the several wards invaded had all been resident in the hospital for thirteen days and upwards before attack; all of them but one for nineteen days or more before they fell sick, a circumstance that seems to indicate that the children did not themselves introduce infectious disease to their several wards. . . . . In Louisa ward scarlatina was followed, after intervals, varying from twelve to eighteen days, by scarlatina, measles, and diphtheria. In Alexandra ward diphtheria was, within three days, followed by cases of scarlatina, and by acute albuminuria. In Alice ward diphtheria was quickly followed by diphtheria and sore throat, and whooping-cough; and, after an interval of nearly three weeks, by an additional case of diphtheria. . . . Thus, Mr. Power finds it necessary to close his report without having arrived at any definite answer to the question how diphtheria was produced in the hospital—a disappointing, but apparently inevitable, result."

† Dr. Low describes a case in which typhoid fever, originating from bad hygienic conditions, was itself propagated by infection. He thinks that, under progressive development, infectiveness may become elaborated from a succession of attacks of diarrhœa.

‡ Surgeon-Major Don.

§ Cuningham. See Macnamara, p. 90:—"As an example of the lengths to which the followers of the specific poison theory have to go in their search for explanation, according to that theory, for the cause of 'enteric

Contamination Theory.—I apologise for giving expression to so simple and elementary a truth as that contamination by foul matters of air, earth, water, meat, or milk are, individually and collectively, causes of disease. But are they so of any one particular form of disease, specific in character, rather than of a generally deteriorated state of body, the result of which presents itself as increased susceptibility of, or liability to, disease generally, and especially to that, whatever its form, which occurs epidemically? Such conditions are seldom unconnected with crowding, personal dirt, hunger, insufficient food; too often, habits of dissipation, vice, crime, —moral, equally with physical, deterioration. Thus, as with the one set so with the other, insanitary conditions naturally lead to, as often they are themselves results of, degeneracy, both bodily and morally.*

Air.—That air polluted by animal matters undergoing decomposition induces diarrhœa, dysentery, in extreme cases what is called putrid fever, and cholera, is within the experience of most observers and writers. That such results do not always necessarily arise from this cause is no less within their cognisance. Factories of preparations the most offensive have remained absolutely free from cholera. When in 1861 that disease more than decimated our troops at Mean Meer, so intense was the peculiar and characteristic cholera odour in hospital and vicinity, that it was perceptible from, and at a distance of, eighty yards; yet the propagation of the disease was not attributed to this circumstance.

If, on the one hand, instances are recorded of the occurrence of yellow fever in connection with emanations from decomposing matters on board ship in latitudes within the sphere of that disease, so instances are on record where, with a decomposing mass of hides on board for weeks together, the mal-odours arising therefrom have blackened the lead paint in the saloons and cabins; have produced such a de-

or typhoid' fever, the following extract, from a letter which recently appeared in a medical journal, sufficiently indicates, viz.:—'From time to time I have had cases of typhoid fever under my care when the local dispositions were anything but favourable for such, nor was I able to trace their origin to a typhoid source. What struck me as rather remarkable, in connection with these cases, was their occurring in houses where persons who were much about them prior to and during their illness had leucorrhœal discharges, and none of which persons got the typhoid. The idea has occurred to me frequently (and no work that I have looked into on the subject alludes to it as an origin of the disease) that perhaps the matter which produces typhoid fever may be, or reside in, some special kind of uterine, or vaginal secretion.'"

* See p. 11, No. 2, *New Series of Reports of the Medical Officer of the Privy Council*, 1874.

gree of congestion of the mucous membrane of the nose and fauces as to persist for many months after landing: and yet, although measles and boils prevailed extensively on board, there was no specific fever among the troops or crew.*

Here is an example of what all the large cities of China are more or less;† and in which, Reports state, "no epidemic of febrile disease came under observation". "Density of population, poverty, filthy condition of houses and streets; it might have been presumed that enteric fever, at all events, would have come to light, seeing that the products, whence its organisms are supposed to be derived and nourished, abound in many directions". "Latrines numerous; constructed without regard to cleanliness; their contents kept for months; the neighbourhood saturated with odours of the most intense description; and yet, in some private houses and restaurants in close vicinity, the people well and lively, carrying on active and prosperous business."

Of Pekin, it is stated, "We are obliged to pass certain localities at all times with closed nostrils, while hundreds of people continually live in and around and above these open cesspools, and yet manage to look well and healthy."‡

According to an eminent writer§ on fever in England, "it has never been demonstrated that any particular gaseous body can induce the lesions found after death from enteric fever".‖

b. Earth.—There is no question as to the general fact that, in India, the prevalence, and frequently, also, the occurrence, of cholera, has a distinct relation to locality, including soil and other special conditions. Localities, the soil of which is impregnated with cholera discharges, are thereby rendered absolutely dangerous as camping grounds. But the relation between cholera and the state of the soil as regards

* Such conditions existed on board the *Marlborough*, homeward bound, in 1856.

† *Customs Reports*, No. 13, part 6, January–March 1872, p. 41. Also No. 5, to 31st March 1873, p. 41.

‡ *Customs Reports*, July—September 1872, p. 41. § Dr. Harley.

‖ The following is taken from the *Globe* newspaper, and refers to Paris, viz.:—"The *Havas Agency* has published a reply to the articles which have been written in the different newspapers respecting the dreadful smells that poison the air of the city. Those who suffer from the pestilential miasma are informed that, notwithstanding the manner in which their olfactory nerves are offended, the death-rate in the city has considerably diminished during the past month." The object of these remarks is to show that offensive odours are of themselves not necessarily a cause of sickness. The circumstance also is suggestive that plumbers and other persons whose work is in connection with drain-pipes, sewers, etc., do not suffer in an especial degree from fever, or from any particular form or type of fever.

conservancy is by no means constant. From 1854 to 1867, cholera was absent from Hurdwar, the state of conservancy meantime the same; in the latter year, the first regular attempt was made to improve the place in that respect, and then the disease recurred with great violence. Filthy places at Lucknow, Morar, Constantinople, Rome, etc., in 1864-5, and similarly in Central India in 1875, escaped; while others, clean, and in better general condition, suffered.

Of Pekin, it is said, "with all our filth, dirt, and smells—and people in the west can form no notion of what they are, for they almost defy description—there is a wonderful immunity even from fevers. The police and scavengers are among the healthiest and most robust of our population. The beggars, a numerous class, sleep in the streets nearly all the year round—to some extent they contest with dogs for refuse of the dunghills—still they survive and flourish, and most of them look fat and sleek".* Similarly, to my personal knowledge in Tientsin, *Hwo-luan* or sporadic, *Wan-yih* or epidemic† cholera, known in China since B.C. 2500, occurred severely in Pekin as an epidemic in 1821 and 1862, and on other occasions. In the intervals, local conditions were precisely as they were during the prevalence of the disease.‡

In India, the term "filth disease" has of late years been theoretically applied§ to fevers of an adynamic type, or with intestinal complication, in a sense intended to explain their etiology, and preventive measures have been applied accordingly. But so far have those measures of conservancy and scavenging, applied to stations, been from reducing the fatality of fevers returned synonymously typhoid or enteric, that, as already detailed before this Society,|| proportionate mortality has increased very notably. Of yellow fever in New Orleans, it is stated that the city was not in a worse condition as regards dirt in 1878, while the epidemic raged, than it was in 1877, or 1876, or 1875, when there was no epidemic; and inferences were drawn on the spot which, if justified, militate against sanitation, according to the simple theory of which the nasty word filth is the expression.

With regard to fever, my printed reports and published articles on the subject contain somewhat lengthy details, in which, by the evidence of capable observers and recorders, outbreaks of fever, presenting characters such as are held to

* *Customs Reports*, July–September 1873, p. 49.
† *Customs Reports*, July–September 1872, p. 41.
§ *Med. Reports, Privy Council*, No. 5, for 1873, p. 9.
|| Notes on Fevers, p. 1.

‡ See p. 39.

distinguish typhoid or enteric, have occurred on virgin soil, where contamination by filth or other organic matter was simply impossible. Among such places are enumerated Natal, New Zealand, Cyprus, Ascension, etc. Even in localities, and on occasions when either or all the diseases named co-exist with "insanitary" conditions, it is well to demarcate between such coincidences and the relation necessarily connecting effect with cause.

On this subject of local contamination, a well-known professor and writer* on Public Health observes that "all who are deeply interested in sanitary reform seem to imbibe an unconscious predilection for the doctrine that all diseases which prevail epidemically are the offspring of filth and overcrowding. This extreme doctrine is rejected by all the best authorities." He then discusses the subjects of contagion and epidemic influences on constitution, furnishing the explanation of phenomena which the theory of local contamination alone fails to demonstrate. A still later writer† takes up the latter subject, and I should have wished to present their views. They can now, however, be no more than thus alluded to. My limits altogether preclude me from following them.‡

c. Water.—The capacity of water to convey the disease cholera, under certain circumstances, is acknowledged. Not so the precise manner in which this takes place, nor the precise nature of the things conveyed, whether germ,§ poison, or morbific influence. According to the International Commissioners, doubts are expressed as to water thus contaminated being rendered innocuous by either filtration or boiling.‖ Chemistry also fails to detect the existence of contaminating material of specific kind. But there are many instances on record where cholera has spread in localities where contamination of water was impossible; also of two sets of troops using water from the same source, the one set

* Mr. James Ball writes thus in the *Journal of the Society of Arts*, January 21st 1881:—"Professors of sanitary matters are so utterly at variance upon what they individually consider to be vital questions, that the public could not place anything like implicit reliance upon the reports furnished them."

† Dr. Guy, *Public Health*, p. 88.

‡ J. Parkin; see *Journal of Science*, November 1880, p. 715.

§ In 1870 Professor Cohn described a peculiar microphyte, *Crenothrix Polypora*, which he found in the well-water of a certain district in Breslau, famous for the frequent occurrence of enteric fever among its inhabitants.

‖ Dr. Parkes stated that "it is uncertain how far boiling will destroy the poison of specific diseases. Purification of river water for drinking by means of boiling was practised in the armies of ancient Persia B.C. 550."

being attacked by cholera, the other set remaining absolutely exempt. Contaminated water induces also other diseases than cholera, more especially diarrhœa and dysentery. It does not of necessity induce cholera, even in places where that disease is endemic, epidemic, or both. In those where it is neither endemic nor epidemic at the time, water, however contaminated, does not induce it.* Many German authorities are opposed to the theory of choleraic water poisoning, on the grounds that it failed to account for the diffusion of the epidemic of Munich, Bavaria, Saxony, Baden, and villages around Vienna.†

To quote one of the most recent writers on this subject‡—"Did we find outbreaks of cholera uniformly occurring at those seasons of the year when the drinking water of the community is most foul we should with reason connect the phenomena. But it is not so. Thus, in Bengal, cholera is on the increase in October and November, when the drinking water is about its best; and the spring outbreaks occur towards the end of February or the beginning of March, at a time when the water of rivers, tanks, and wells is as yet unpolluted by the rain washings of accumulated dirt from the surrounding surface."

With regard to fever in India, there is a concurrence of opinion that in malarial localities attacks of malarial fever may be, and are, induced by means of water in such places, contaminated with decomposing vegetable matters. As explained at considerable length in my Reports and printed Articles on the subject, cases of malarial fever become in their progress complicated with infiltration or ulceration of the glands of the ileum, as with other forms of organic lesion; the phenomena of the disease in particular individuals and stages of attack assume a low, adynamic, typhous, or typhoid character. In this sense, there appears to be no doubt as to the influence of contaminated water in the causation of fever. But, as a matter of fact, of 175 cases,

* On this subject Dr. Parkes said, "although the origin of typhoid merely from putrefying non-typhoid sewage is not at present considered to be probable it is not disproved."

† At Newcastle in 1854, and in reference to the now famous Broad Street pump case, it was not until cholera had become epidemic in England that contaminated water induced an attack of the disease.

Dr. Somerville observes in his *Report on the Health State of Foochow*, for the half-year ending 30th September 1875—"I think there is a growing opinion in the profession, especially entertained by those who have had much experience in tropical and sub-tropical countries, that water containing organic matter has been too heavily blamed as a cause of disease."—See *Customs Reports*, No. 27, July–September 1875, p. 43.

‡ Macnamara, *Himalayan India*, p. 118.

details of which are given in my first special report, and 10 additional contained in my second, in not one instance was a connection established between an attack of fever and the water made use of by the subject of attack.

Here is an illustrative instance of what is looked upon as water causation recently adduced in India. At a particular station* twenty-eight cases of fever occurred among soldiers; of these, twenty-four in a body of teetotallers, who were quartered in various localities in that station. The men of abstinence were wont to assemble at their place of meeting, and there, in the words of the reporter, make merry on their beverage drawn from a pump hard by, the pump unused by other men in the cantonment. And so, at the risk of causing alarm to the class referred to, the cause of illness was assigned to the cause of water only, all other circumstances and conditions affecting the men in question being passed over silently. But what of the cases to which this cause could not be assigned? What of other men of the class alluded to, their number left unstated, who had no fever? That persons whose social condition, habits, and mode of life present similarity should be subject to prevailing diseases of allied or similar type is but in accordance with analogy. That the class here indicated is more likely to suffer from fever, asthenic in type, than from ardent or inflammatory, is in accordance with observation.† But as a development of the theory of single, and it water, causation here alluded to, I remark that trustworthy statistics are wanting as to comparative coincidences between cases of fever and particular sources of water supply in affected localities, and coincidences between fever attacks and supplies from butchers, bakers, grocers, or other establishments in the same localities. In India, all these sources of supply are equally under strict supervision. Also, has the oxidising power of water upon the decomposing organic matters, recognised only a few years ago, been now clean forgotten? Do any of my contemporaries recollect their experiences of outward bound voyages in sailing ships ere the first half of this century had yet been reckoned with the past? Do they recollect how the water casks in those days had the reputation of being filled from the Thames at Blackwall while the tide was low; that afterwards, while the process of what was called "settling itself" was in progress, foul odours of sulphuretted hydrogen rose with violence from the casks; then after an interval this

* Kamptee.

† Compare this with the remarks already quoted from Surgeon-Major Don regarding the classes most liable to asthenic fevers in Bermuda.

"settled" water became sweet and palatable? And do they also recollect the wonderful measure of health enjoyed on board such vessels, no fever, no bowel disease of any consequence, no deaths among a strength numbering close upon 200, during a voyage ranging in length from four to six months, their numbers greater when disembarked than when embarked, by reason of births on board?

This is the state of the water-supply at Canton, as described not long since.* "The creek San-t'sung is not far from the foreign settlement; it is narrow, and covered with boats; on either side of it are innumerable houses, chiefly brothels; the alvine dejections, and other impurities of thousands of inhabitants along it, are daily discharged into the stream; yet the water, too dirty even for washing, is daily used for culinary purposes without being filtered or precipitated with alum, as is done in Shanghai."† Here we should expect the prevalence of such diseases as typhoid fever and diarrhœa

* *Chinese Customs Reports*, No. 13, part 6, January-March, 1872, p. 21.

† "At Shanghai, the creeks receive drainage from the manured fields, and in them the natives regularly scour their ordure buckets. From such a source is the water-supply for foreigners obtained."—*Customs Reports*, July-September 1873, p. 55. It is true that the Chinese, as a class, drink only water that has been boiled, or in which tea has been infused, and that their relative exemption from cholera has been attributed to this circumstance. See references already made to the assumed resistance of theoretical specific poisons to the process of boiling. In all shapes such causes of pythogenic fever, as are usually assigned, exist abundantly in Chinese towns, yet only isolated and rare cases presenting characters of that disease are reported among the dense population, nor has this form, apparently as yet, spread as an epidemic. It were strange if among the many characters assumed by the general disease fever, particular lesions in the intestines, as of other organs, did not occasionally happen. There is, however, nothing in that circumstance to indicate that in their nature or causation they differed from those, the complications of which affected other organs and tissues."—Dr. Gardener, quoted in first special report, p. 11. At p. 21 of the *Chinese Customs Gazette*, January-March 1872, the following occurs in reference to typhoid fever: "Since the sewage question has been so much in agitation in connection with this fever, it may be well to mention that in Canton large numbers of the native population are daily using water, and inhaling air charged with the impurities of human excreta, apparently with utter impunity. River water is greatly used, but that used by the crowded boat population along the different jetties is extremely filthy. The Sampan people prefer to get it by the side of their own boats, simply because they receive no harm by the practice." Nothing appears to indicate other than that among the boat population of Canton, water is used in the ordinary way, that is, boiled for culinary purposes, unboiled for other purposes. This significant remark occurs, however, at the place quoted: "The natives have no faith in the skill of foreign physicians in the cure of fever, and when taken with it they do not send for them, nor do they come to the hospital to be treated as in-door patients. From native books and physicians we can gather no distinct idea of such a disease as typhoid fever among the Chinese."

occurring often enough to attract attention; but, according to the observer* from whom I quote, the people who use this water are not more subject to fevers and other diseases than their neighbours, and this impunity is one of the reasons for their continuance in the use of such water. The writer quoted remarks in reference to this circumstance: "A detailed examination of this creek, and the disgusting habits of the inhabitants, would almost unsettle one's idea of the connection between typhoid fever and polluted water." At Foochow,† conditions are described as equally bad in this respect as at Canton, and the same may be said of many other Chinese towns and cities, and yet there is no enteric fever in them, and, for the most part, cholera, when it does occur, is, by accounts, less fatal than it is in India or in Europe.‡

As a contrast, I quote thus with regard to cases, said to have been of enteric fever, at Ascension.§ "The water-supply, rain water, spring water, to which all contamination is prevented, and condensed water; all three waters kept stored in stone and iron tanks, and cannot by any possibility be exposed to sewage contamination; and yet nine cases of 'enteric' fever are said to have occurred on that island; the characteristic symptoms of that disease present in all its varying degrees. No material impurity in the water or tanks; no fault with the hygienic state of the garrison, or of the farm establishment at Green Mountain."

Food and Milk.—In certain Eastern nations, particular articles of food are, by means of religious and other enactments, declared unwholesome; in other countries, those articles are in common use without evil result. In India, the use of milk is general, many sects of natives consuming it in great quantities. These classes are careful, however, that the milk so used by them is in the first instance boiled, coagulated, and otherwise prepared. Experience on their part, and on that of Europeans in that country, indicates that in an unprepared state, but particularly in places where cholera is endemic, or where it is epidemic, there is considerable danger attending the free use of this product.

* Dr. F. Wong.

† *Customs Reports*, January–March 1873, p. 41.

‡ A similar state of things is described as existing in Japan. "There, as in China, the fields and growing vegetables are at short intervals of time drenched with liquid human filth; the canals and watercourses are contaminated by water which escapes from the fields, and yet no special disease among the population is assigned to this cause."—See *Journal d'Hygiène*, No. 228, of 3rd February 1881.

§ *Health Report of Navy*, 1879, p. 54.

But there is no apparent reason to assume that the actual source of such danger is anything exterior to the changes which spontaneously take place in the fluid as a result of general conditions prevailing in that particular place. In China, milk is looked upon as an excretion, and abhorred as unclean. No doubt there are sanitary reasons for its being so. In India, instances have occurred in which there could be no reasonable doubt that cholera had been induced by the use of milk containing water contaminated by cholera dejections. But, on the other hand, infants fed at the breast by nurses ill with cholera, if in some instances they have been themselves attacked, they have escaped in others. As a general result, milk contaminated by cholera matters is most dangerous to individuals partaking of it.

With regard to fever, only such as is attended by ulceration of the small intestines has been attributed to contaminated articles of food, including milk. How stand the facts in reference to this point? In no single instance of the somewhat numerous cases investigated by me in India was a connection traced between the occurrence of fever said to be enteric or typhoid, and the use of contaminated or deteriorated meat. Indeed, no such meat was made use of either by soldiers or their families. As to instances in which elsewhere the occurrence of typhoid fever has been attributed to this cause, the disease thus induced has subsequently been referred to trichinosis, and then to sausage poisoning. On this point, I refer to remarks already made in this paper under the head of *Specific Origin Theory*.

I am aware that cases, said to have been of enteric fever, have by competent writers* been attributed to eating tainted tinned meat, the circumstance assigned theoretically to the development of an assumed poison, putrogen. Also that there are those who believe that eating the flesh of an animal affected with typhoid fever can bring on the disease in the person who eats it. Compare these remarks with those already made, and then with the following:—

At Shanghai, in 1872, rinderpest prevailed extensively among the cattle. Beef of animals thus affected was extensively eaten by the sick inhabitants; no evil result followed. But, as expressed by the reporter, "it is absurd to recommend people not to be alarmed, when the meat which supplies their tables may have been cut from animals dead of a disease which from *post mortem* appearances might be described as a combination of diphtheria, typhoid fever, and dysentery."† In India, the Kotahs, a Dravidian tribe inha-

* *Dr. Lowe.* † Customs Reports, January–March 1872, p. 79.

biting the Neilgherie hills, habitually eat carrion, and in Bengal, certain wandering mendicants devour dead matters of the most horrible kind,* in both cases without any such result as might be looked for to accrue. I purposely refrain from prosecuting this part of my subject, as it refers to England. It is on record† that in Paris people have inoculated themselves from animals suffering from cattle plague, have eaten the flesh of those affected with that form of disease, and even of those affected with carbuncle, without evil result. It is, moreover, stated on authority that the cooked flesh of those suffering from typhus is not unfit for food. According to a paper submitted to the Paris Academy of Science, although milk from cows suffering from typhus is unwholesome, in other respects "neither it nor meat are capable of transmitting typhus to man or any animal except a ruminant.‡ As to the generally pernicious and most objectionable qualities of contaminated meat and milk, there is no question. But we are dealing with specific diseases, and them only.§

10. *Youth and Recent Arrival.*—As a principle, all ages are equally liable to attack by cholera during epidemics of that disease. As a second principle, the recently-arrived European, of whatever nationality, is more liable to be attacked than those of longer residence. Exceptions to this occur, however; notably in the outbreak of cholera among the cavalry at Secunderabad in 1871. On that occasion, the men first attacked were those of longest residence in India, and there are other instances of the same kind. But these exceptions are not sufficient to affect the general rule as expressed.

With regard to fever in India and the tropics generally, circumstances are somewhat different. In all, sickness, especially by the several forms of fever, but particularly such as affect the intestinal canal, attacks the young and recent arrival to an extent proportionally greater than it does the man of more mature years and longer residence. Inasmuch as in temperate climates seasonal conditions which prevail in autumn induce bowel complaints in various forms, so in the

* *Medical Jurisprudence for India.* By Dr. Chevers.

† *Hygiene and Surgery of Franco-Prussian War*, p. 225.

‡ Id. p. 226.

§ Dr. R. B. Lowe has been told that rabbits and cats suffer from a disease in all respects like typhoid. He says it is just possible that some case may have originated from eating the diseased rabbit flesh, and that rabbits may have become infected by contagion from the discharges of a typhoid patient. Beaugrand gives the case of typhoid fever caused by eating the flesh of a wild kid caught in a snare. He has ascribed diarrhœa, and other illnesses, to eating rabbits.

tropics corresponding seasonal conditions produce similar results, whether as independent affections or as complications of others. Youth and adolescence are the periods during which deposits in, and other affections of, glands and gland-like tissues are of most frequent occurrence. Hence, doubtless, the occurrence of infiltration, and in some cases of ulceration, in the glands of Peyer in other diseases than fever, and in some instances where during life there was no apparent illness whatever. This circumstance has in India presented itself in cases where death by heat apoplexy occurred within a couple of days after attack. Such a state of glands appears to me, like scrofula, to be properly indicated as a condition. But where the condition exists, there constitutional disease, however induced, will naturally affect tissues thus impaired, nor is the introduction of a specific poison required to bring about that result. Young soldiers are more impressionable to all forms of endemic disease than old. They are also more indiscreet and inexperienced as regards exposure to well-known causes of illness.

11. *Races.*—In some epidemics of cholera the natives of India suffer more severely by that disease than do foreign residents; in others, they suffer less. Aborigines of the hilly districts of Nagpore are said to be exempt from cholera so long as they remain in their own native localities, but to be peculiarly prone to that disease when they enter the plains. A similar circumstance has been observed in reference to Ghoorkas from Nepaul; and it is further on record that during the epidemic of 1869 in Persia scarcely a negro escaped. In that of 1877-8 in Southern India the disease affected Europeans to a small extent compared to what it did the natives. And yet, with a very few exceptions, cholera has in turn affected all nations and nationalties. This remark applies also to small-pox.*

On the coast of Guinea the native African is to a great degree exempt from fever, of which the European visitor is nearly certain, sooner or later, to be the subject, and very

* In China a large proportion of European mothers are unable to suckle children. Small-pox and syphilis, when attacking races for the first time, are characterised by a virulence and deadliness seldom met with in their subsequent history. An extension of the same principle will help to explain the decay of epidemics, or the complete extinction of some diseases.—*Chinese Customs Reports*, July–September 1873, p. 31. "The acquisition of a high degree of civilisation, humanity, and science, tends to the fostering and propagation of forms less able to resist disease on account of its endeavouring to preserve, and its success in fostering the weak and susceptible. This is a drawback to civilisation."—*Op. cit.*, *p. 32.* This picture can scarcely be said to be complimentary to modern *hygiene*.

often the victim. The native races are altogether exempt from those outbreaks of fatal epidemics which, from time to time, have made havoc in ships' crews in the estuaries of those great rivers which occur along the West Coast from the Gambia to the Gaboon. In India it is stated that Non-Aryan races in Assam suffer to a comparatively greater extent from "malarial" disease than do Aryans in the same provinces. On the other hand, a race of people, viz., the Tharoos, Aryans by race, withstand the pestilential climate of the Oude *terai*, which is deadly to all others.* In China, the native of the country and the European appear to suffer equally from malarial fever, and from that form designated typho-malarial.†

Natives of India and Europeans of various nationalities suffer from altogether different diseases, and variously from the same diseases. This rule holds good to such an extent as affecting the very existence of the British as permanent settlers in, and colonists of, that country, that so far as I am aware the assertion of a very eminent medical officer has never yet been controverted, viz., that a third generation is wanting of pure Europeans, resident continuously in India, and without admixture of new or imported "blood".‡ Thus, by analogy, we are led to what is demonstrated by actualities, viz., that the phenomena of disease differ according to race. This is so to a marked degree in the case of fever. But of this again, under the head of climate.

12. *Individual Conditions.*—Although delicate physique and ill-health predispose to cholera, and persons sick in hospital manifest a great liability to attack and death by that disease when epidemic, yet exceptions are so frequent that a rule on this subject is far from absolute.

With regard to tropical fevers, circumstances are more defined. Thus, according to a recognised authority§ on this subject "the severe, or ardent fever, called also climatic, or

* *Tharoos*, believed to have been originally from Chittore, whence they escaped on the capture of that place by Akbar about A.D. 1560. They claim to have been originally Rajpoots; that their ancestors lost their caste by drinking intoxicating liquors, and rearing fowls. The present descendants are much given to the vice of drunkenness. They keep their houses scrupulously clean. They drink water from wells only.—Sir John Elliott's *Races of India*, vol. i, pp. 317-18.

† From the *Customs' Reports*, July-September, 1872, p. 59.

‡ This question of relation of race and disease presents considerable difficulties. While in China the native Chinese suffer from malarial fevers to quite an equal extent as Europeans (*Customs Reports*, July-September 1872, p. 35), the natives of India in many instances, similarly, suffer more than Europeans. On the other hand, there is reason to believe that the Jews in England are more healthy and long-lived than the English people are.

§ Copland.

seasoning fever, occurs among the recently arrived,—and more especially among the young, the intemperate, the robust and plethoric, and among those exposed to the sun in very high temperatures." According to the views formerly held in India, fever occurring in such subjects, sthenic in its earlier stages, in its later became complicated with visceral derangement, intestinal and otherwise, attended by a low, typhoid, or adynamic state, in persons of weakly frame, asthenic from the first. Of fever in Bermuda, a similar observation occurs, viz., that "pathological changes depend upon contingent circumstances, personal and otherwise, that enteric fever (that is, fever with intestinal complication?) invariably occurred in persons of feeble muscular development; while it was observed that the simple continued fevers without bowel lesion, were generally in persons of more robust frame." On the other hand, physical conditions, manner of life and habits, whether natural or acquired, may be modified as to predispose to adynamic forms of disease, and to determination to particular viscera.

13. *Climatic and Seasonal Influences.*—Under the operation of the sum of conditions briefly expressed as climatic, differences, specific and generic, occur in all branches of the great organic kingdom met with in the several zones; often, also, in different parts of the same zone. That a few, extremely few exceptions which occur, but tend to prove this rule is doubtless true; but the rule is as stated. And so, also, with disease. As with growth and development, so with decay, and, often, the manner in which this takes place: all of these are affected by conditions, geographical and climatorial. Hence, arises the general question of geographical distribution of disease.*

As in temperate climates, the process of growth, development, and subsequent disintegration are, for the most part, in progress slow, so in tropical are they rapid. If, in some instances, animals and plants undergo, more or less completely,

* In what may be called the Western tropics, the modifications of fever are thus noticed by a very distinguished member of this Society. "Cases commence as remittent, and continued as such from the sixth to the tenth day, on the same afternoon the fever recurred, rapidly took on the character of yellow fever, and proved fatal on the fourth or fifth day of that form. In other cases, which commenced as intermittent, diarrhœa ensued, after three or more tertian periods the fever became continued, assumed the character of typhoid, ultimately presented the affection of the kidneys and urine seen in yellow fever, then terminated in death. The intermixture of morbid appearances, peculiar to yellow and typhoid fevers, was detected in variable proportions." Inspector-General Lawson, *Medico-Chirurg. Review*, July 1869, p. 225, printed in *Chinese Customs Reports*, part 6, July–September 1872, p. 59.

a process of acclimatisation when introduced from one climate to one nearly similar, the great majority refuse to do so; while of those introduced from a climate of particular character to one altogether different, many perish; and those that continue become modified in various ways, lose the power of reproduction, or their descendants rapidly degenerate. And so with regard to manifestations of disease, the nature of attack, the organs most liable to suffer, differ under one set of climatorial conditions from what they are in another.

On this section of my subject, I summarise conclusions expressed by a competent and recognised authority.* "The primary effect of great changes of climate is on the circulation; the blood drawn surface-ward by heat, driven in by cold. The secondary effect is increased external vascularity during heat, internal during cold. In the tropics the vascularity of adult lungs is reduced, so also are their spirometric measurement, their functions, the nephritic vascularity and secretion; those of the skin are increased, the circulation becomes more languid, the pulse less frequent, and less forcible; the organs and functions of animal, like those of vegetable life, are affected by great changes of climate, heat impairing the weight, strength, and health at all ages, and retarding growth in youth." The balance of conditions and functions, here indicated, being more or less completely and suddenly deranged, disease becomes a natural result. It so occurs; its type and incidence, as regards organs and tissues, influenced by conditions already stated.

Those who, depressed, worn out by endemic disease, or by simple exhaustion incidental to a tropical climate, say of Bengal, as an example, speedily realise the benefits of a trip to sea, or to the bracing atmosphere of the Himalayas. So do they alas! and very often conversely, when scarcely half recovered, duty forces their return to localities in which their health gave way. To such, and they are many, the expression "climate" indicates a very stern reality.†

A very eminent authority‡ writes on the same subject

* Dr. Rattray. Paper before Royal Society.

† "It is a common observation with men, when discussing the unhealthiness of these climates (China), that their evil reputation is not deserved; that they are as healthy as our native lands, and that we should find them so, did we only assimilate our diet and habits to those of the natives." "There is a sophistry about this which deceives some, and others like it, because they belong to a morbid class, who have a natural tendency to do anything entailing a self-denial, who think that whatever is pleasant must be bad, and whatever is unpleasant, and involving self-denial, must be good."—*Chinese Customs Reports*, July–September 1873, p. 32.

‡ Dr. Guy, Public Health, p. 31.

thus: "Every year we have some atmospheric element, which neither thermometer nor barometer, neither rain nor wind-gauge, nor measure of moisture, nor test of ozone, can reveal to us, but only our records of sickness and death. One year it is such as favours small-pox; the next, perhaps, it will promote scarlet fever or measles, or whooping-cough; or it will, so to speak, select from several forms of fever that one which shall fill the beds of our fever hospitals. The condition of air, which disease itself is the only test and measure, was once called pestilence, but is now known as its epidemic constitution — *la disposition atmosphérique génératrice.*"

14. *Contagion* plays relatively an inconsiderable part in regard to cholera and fever in the tropics. With regard to the former disease, while there are circumstances which support the theory of transmission by this means in respect to individuals and communities, there are other, and equally numerous, which point to an opposite direction; the result being, that in one set of instances it is beyond question that the disease was thus transmitted; in other instances, it is equally indubitable that it was not so communicated.

It has been pointed out* with regard to a succession of outbreaks of cholera in India, that in places where the epidemic was most intense, the disease was often confined to very circumscribed localities, and points of immunity are mixed up with those which suffered all over the country to a varying extent in different districts; also that in the affected and non-affected localities the conditions of the people were alike, and free communication went on among them.†

As a matter of fact, it is within the experience of many observers in India that in times of cholera a general tendency to bowel affections prevails; that in other respects the health standard of masses is impaired, and that during the continuance of such conditions, depressants of all kinds, medical, physical, or moral, are to be avoided. Exceptions which occur do not alter the rule as it stands. The recurrence of cholera periods is familiarly recognised as a cholera wave. There are also fever years, unhealthy years, during

* In the last report by the sanitary commissioner with the Government of India, viz., for 1879, Dr. Cunningham expresses strongly his views that neither propagation by men, that is, contagion, nor fouling of water by cholera discharges, can account for the diffusion of cholera, *Medical Times and Gazette*, February 5th, 1881. It is desirable to bear in mind the difference between the communication of cholera in isolated cases, and the general diffusion of that disease as an epidemic.

† Paper by Inspector-General Lawson. See *Transactions of the Epidemiological Society*, 1879, p. 91.

which diseases, of whatever nature, external or internal, assume severe or peculiar types. All these phenomena pointing to the operation of a cause above and beyond a specific undiscoverable entity, transcending, but it may be, including the latter.

The theory of pandemic waves merits careful attention, for the reason, on the one hand, that it appears to be but another expression for epidemic influence; on the other, on account of the high position as an investigator, enjoyed by its leading advocate. According to him, its nature and mode of action are difficult to define; it may act by rendering the system more susceptible of the ordinary causes of disease, or it may act upon those causes themselves, giving origin to a more virulent, or new form of disease.

The existence of contagious fevers* in India is placed beyond doubt. Such a fever has been described in 1815 and 1820 in Kattywar, Kutch, and Guzerat;† more recently the occurrence of malarial fevers as epidemics has been recorded as happening at intervals of ten years,‡ the circumstance of the disease on such occasions being contagious or not, being much debated. Then comes the Pali plague, the Maha-Murree, the great sickness§ in 1834-5, 1849, 1851-2, 1860, 1876-7, its contagiousness apparently limited to race. But with regard to the ordinary fevers of India, such as came under my notice, out of 175 cases investigated, not one was traced to either contagion or infection.

15. *Summary.*—In summarising the remarks now made, the following points present themselves when viewed from the aspect from which I consider the data thus examined. The distribution of the diseases named in different portions of India, on the occasions described, is to be accounted for neither by the theory of specific poisoning alone, nor by that of contagion alone. There is every reason for believing that, although no actual relationship exists between the diseases, cholera and small-pox, the same, or a similar law applies to

* With regard to what is called enteric or typhoid fever, Dr. Murchison says that hospital experience lends little support to the doctrine of contagion. Dr. Wilks had never seen a case where it was contagious among the nurses of Guy's Hospital, nor had Dr. Peacock in those of St. Thomas's Hospital. Messrs. Bristowe and Holmes had seen two cases in which it was contagious in the Royal Free Hospital. Dr. Collie is of opinion that it is contagious. Dr. Sharkey believes contagion to be more frequent as a cause than is supposed.

Dr. Chevers gives, *Transactions of the Epidemiological Society*, vol. iv, part iii, p. 338, particulars of an epidemic of relapsing fever in Yusufzye in 1852-3, of violent remittent in Upper Provinces in 1816; in other parts of India in 1836-7, 1855, 1860, 1864.

† *First Special Report*, p. 8. ‡ Macnamara, p. 301.

§ See p. 320.

the manner of their prevalence as epidemics. Between cholera and malarial fevers a relationship is indicated as existing, alike in respect to causation and phenomena. Both these diseases in India present a general relation to season. Both in India and England a relation appears to exist between the type of prevailing fever and locality. Bowel affections generally, in India and elsewhere, present a relation to season and locality; this relation as defined as that of vegetation to the same circumstances. In endemic fevers intestinal lesions, and those of the abdominal tissues, occur as complications in protracted cases. Cholera in India occurs under a variety of circumstances, and in different manners, but neither furnish a specific name applied to particular cases, as indicating one particular manner of causation. Fevers vary in phenomena and intensity, according to conditions, geographical, climatic, and individual. Among our soldiers in India fevers in the mass, and in individual cases, are less sthenic in type than they formerly were. Reasons for this circumstance are assigned. Specific poisons are of two categories, as described. Innocuous liquids can, by artificial means, become converted into poisons; hence, analogy indicates the likelihood of corresponding changes being effected by means of vital action. But diseased liquids do not necessarily communicate their particular disease, either when inoculated, or when swallowed. Certain diseases named, which are propagated by infection or contagion, are induced by particular combinations of conditions. Fever, in some of its types and forms is of this class; so is cholera. Between these diseases themselves, and with regard to several others named as occurring epidemically, an affinity is indicated. The expression malaria is but another term for climatic influences. The existence of organisms peculiar to malaria has yet to be confirmed. No specific poison is necessary to account for the phenomena of Indian fevers. The theory of specific poison does not explain those in "typhoid" or "enteric" fever. It is opposed as insufficient by a number of authorities. General insanitary conditions predispose to disease, but do they to any one specific disease more than another? It is not established that such is the case. Polluted air induces certain diseases, but not necessarily a specific form of fever. In China air intensely polluted is compatible with health and activity. The theory of local pollution, if sufficient to account for the occurrence of some epidemics, is altogether insufficient in respect to others. Although contaminated water may induce cholera, the occurrence of that disease is not always thus accounted for. Water induces cholera after it has become epidemic, not be-

fore. The relation of water-supply to cholera in India is not established by recent observations. Very striking instances are adduced where the use of extremely foul water has not resulted in fever, enteric or other; while instances are recorded, particularly in places where cholera is endemic or epidemic, of cases of that disease following the use of contaminated milk; instances are related of infants suckled by women ill of cholera, and yet not becoming affected by it. Cases said to have been typhoid fever, due to the use of diseased meat, were subsequently stated to have been cases of trichinosis and sausage-poisoning. Although specific typhoid, like several other forms of fever, may be conveyed by means of milk, this circumstance by itself fails to explain the epidemic diffusion of that disease. As a rule, cholera has no relation to age. In India and the tropics generally the young and the lately-arrived suffer more by endemic diseases than the older in age and residence. Race appears to affect liability to certain special diseases in particular countries; not so in others. Infant mortality of Europeans in India is excessive. Individual conditions and habits predispose to particular forms of disease and complications. Climate influences the geographical distribution of disease, and determines its type and incidence. Although cholera has undoubtedly been propagated by means of masses of persons and individuals in numerous instances, there are others equally numerous in which it has not been so.* Fevers of particular kinds in India may be propagated by means of infection, but of the cases investigated by me not one was thus accounted for.†

Thus, by the light I possess, have I endeavoured to pass in review, some out of many points which, to me, appear most important in reference to the occurrence and prevalence of the diseases named. I am aware that I have the great misfortune to entertain an interpretation of phenomena and evidence before me, different, in several respects, from that of some very eminent authorities. I am also aware that in my manner of interpreting those phenomena, I am in accord with

* *Hygiene of Cholera*, par. 11, p. 99; also pp. 150-159.

† As expressed by the older authors, so now—"Neither infection, nor terrestrial malaria, nor mineral vapours, nor exhalations from dead animal matter, nor intemperature of season or weather, nor famine, scarcity, or unwholesome food, nor crowding of the living—the healthy or sick—nor filth, nor stillness, humidity, warmth, or other conditions of the air, nor depressing emotions and passions, nor any physical, social, or moral vicissitude, will singly account for epidemics; but that the association of several of these causes in various grades of predominance is necessary to their occurrence, diffusion, and continuance."—Copland's *Dictionary of Medicine*, 1858, p. 773.

other eminent authorities and most careful observers; the numbers of both decidedly on the increase, not alone in this country, but more especially in India, China, and elsewhere, among our vast foreign possessions and settlements.

I have restricted my remarks to opinions only. I, however, give references in the shape of foot-notes, the better to facilitate access to the sources of these opinions. I trust that in the paper now read I have, to some extent, at least, succeeded in my endeavour to indicate the grounds upon which views expressed by me have been formed, as also the process by which I have myself been led to consider from the data before me, that the theories of specific poison and contagion are, of themselves, insufficient to account for the phenomena of the diseases to which I have had the honour to solicit your attention, and that of the Epidemiological Society.

ON CERTAIN POINTS IN CONNECTION WITH MALARIAL FEVERS IN INDIA.

By E. G. RUSSELL, M.B., B.Sc. Lond.

(*Read: April 13th*, 1881.)

I have the honour of appearing before the Society this evening in order to make a few remarks on Indian fevers. It will suggest itself to you that such a comprehensive subject as Indian fevers could be touched on only in the slightest and most superficial manner, in the brief space accorded me. Instead, therefore, of endeavouring to wander discursively through the wide domain of Indian fevers generally, I shall attempt to limit my remarks to some few, select, points in the history of what is *par excellence* understood as "fever" in India, viz., malarial, or, as it is sometimes termed, climatic fever. I shall further endeavour to emphasise some of the difficulties in the way of diagnosis of certain other diseases in India—difficulties introduced by the peculiar impress of periodicity which the malarial poison confers on other affections, and pyrexias occurring in the regions of its prevalence. I purpose also making a few brief remarks on typhoid in India, its close resemblance to, and its difficulty of differentiation from, malarial remittent; and, in conclusion, to comment on the occurrence of outbreaks of a fever partaking of the nature of typhoid and of malarial fever, variously termed typho-malarial, or paludo-enteric fever.

I should first desire to invite attention to the complete difference of significance popularly attached to the term "fever" in England, and to that term in India. In England the term "fever" has an ominous ring in the popular ear, and would be commonly understood to suggest some severe, long continued, and dangerous malady, involving serious penalties, such as interruption of social intercourse, isolation of the sufferer, or of the household, removal to hospital—would, indeed, suggest many dreaded potentialities. In fact the term fever would, in England, be taken to mean one of the specific febrile diseases—typhus, typhoid, scarlatina, etc.

In India, on the contrary, the term "fever" is in common and everyday use; but the suggestion to any Indian ear con-

veyed by this term is merely that of an ague attack, or, rather, its hot stage. To a resident in India the bare term "fever" carries none of the ominous meaning that it does to a resident in England, and would, ordinarily, be understood as suggesting nothing more than the hot stage of an ague, or an ague fit consisting chiefly of a hot stage. In, perhaps, four cases out of five, the saying that a person had "fever" would mean that for an hour or two in the day he had an unpleasant flush of heat, with or without a cold or sweating stage, which would lead him to sit in an easy chair, or lie down for an hour or two, and would cause some discomfort; but you would, by no means, necessarily expect him to be absent from his ordinary work, or from the evening muster at the band or badminton party, or to be under any further medical *régime* than the self administration of quinine.

I attach great importance to a clear recognition of the totally different significance which the bare term "fever" has to the mind of a resident in England and to the mind of a resident in India, because I have, on more than one occasion (once, indeed, at a meeting of this Society),seen serious misconception arise from this use of the term "fever" in an undefined general sense.

In my brief remarks this evening it shall be my care to avoid all use of the general term "fever", and to permit this easily-misconstrued little word rarely to appear except in the safe custody of some distinctly defining term, such as malarial, remittent, enteric, etc. Of the two types which malarial fevers may assume, viz., intermittent, or remittent, the former is, I think, in India, as in temperate climates, by far the most common in occurrence. I observe, however, that Dr. Bristowe (*Medicine*, 2nd edition, 1878), notes on p. 285, "remittent fever, the form of ague most common in tropical climates". If by this is meant that remittent is more common than intermittent in tropical countries, I think that the statistics of Indian fevers will point to an opposite conclusion with regard to that tropical country.

My own experience, derived from thermometric observations of some hundreds of cases during nearly seven years' practice in a notoriously malarial region, leads me to the conclusion that, there at least, by far the greater number of cases of malarial fever (probably nine out of ten) were of the intermittent type, by which I mean that the febrile paroxysms were separated by intermissions of complete apyrexia, and that the disease was altogether of a less serious nature. One point worthy of notice is the modification which the form assumed by the disease often undergoes in persons who *have long* resided in malarious regions.

During the first few years of a person's residence in India, and in his first few attacks of intermittent fever, the disease usually assumes one of the typical forms described in text-books. After a long residence, however, in malarious regions, especially if, as is commonly the case, the individual has suffered from repeated attacks of ague, or is tainted with the malarial cachexia, it is extremely common to find intermittent fever assuming the form of a short hot stage only, usually neither severe nor long continued, often merely a flush of heat, and feeling of malaise lasting for one or two hours, during which the temperature rises to 100° Fahr., or 102° Fahr., there being no distinct cold stage, and only an indistinct sweating stage.

An old resident will probably attach little importance to such attacks, and will scarcely vary his ordinary habits beyond the taking of doses of quinine; he will, moreover, look for such an attack of (so termed) "fever" to result from any over-excitement, over-exertion, injury, chill, or, indeed, any marked break in his ordinary habits of life. So well recognised is the frequency of this form of intermittent fever, that the term "fever", and the term "fever and ague", are familiar in our mouths as household words in India; these two terms being used in contradistinction the one from the other; the term "fever and ague" being popularly used among non-medical persons to mean that the hot stage, popularly defined as "fever", was preceded by a distinct cold stage (the popular *sine quâ non* of "ague"); on the other hand, the malarial cachectic, whose attack consisted mainly of a hot stage only, would probably describe himself as suffering from "fever", not "fever and ague", nor "ague", and would expect to be, and, indeed, would be, generally understood in so expressing himself.

Another point of great interest in malarial affections is their periodicity. Periodicity is noted as one of the most marked characteristics of all affections due to the poison of malaria. The malarial poison, moreover, as is well known, not only causes its fevers to assume this character, but, in a person impregnated with the poison, stamps an impress of periodicity on any affection from which he may suffer while living in a malarious region; and this impress of periodicity once acquired seems, in many cases, never to be entirely lost, even after long residence in non-malarious temperate climates.

This term "periodicity", so commonly employed in defining the characteristic feature of malarial fevers, needs to be used in a restricted, exact, sense if it is to be of any ac-

curate diagnostic value in the description of fevers, especially malarious in their origin. On examination, it will be found that in nearly all the diseases accompanied by pyrexia, to which the human system is liable, this pyrexia has periods, often more or less definite and regular, of exacerbation and remission. This may be premised not only of the specific febrile diseases, but also of the pyrexias of local inflammations such as pneumonia, phthisis, etc.; and in both these classes of diseases this periodicity is an ordinary accompaniment in climates where malaria does not exist, and in cases in which no previous exposure to malaria can be suspected—where, therefore, the influence of malaria is absolutely eliminated as a cause. The pyrexia of true non-malarious typhoid often so closely resembles that of true malarial remittent fever that, in India, the diagnosis between these two diseases is frequently extremely difficult, and must be decided almost entirely by other distinctions than the types of their pyrexias. Most medical men in India will have met with cases in which it has been almost impossible to refer the disease with certainty to the one or the other class.

With reference to the periodicity assumed by the pyrexia of typhoid, Professor J. Harley observes (R. Reynolds' *Medicine*, vol. i, p. 389): "One of the most general facts observed in reference to enteric fever is the frequent occurrence of intermittence in the pyrexial condition."

De Claubry notes that the Commissioners appointed to investigate the French epidemics of enteric fever "call attention to the fact that a more or less pernicious intermittent, or at least remittent, character was manifested under a great variety of circumstances". (*Mémoires de l'Académie de Médicine*, tome xiv, p. 71.) On p. 11 of the same work, the same fact is recorded in another form.

Trousseau (*Clinique Médicale*, 2nd edition, p. 247) notes that "enteric fever may simulate at first intermittent fever, and, reciprocally, an intermittent fever may assume at the commencement the characters of typhoid".

Of the typho-malarial, or paludo-enteric fever I shall speak further on; at present I am referring to the periodicity of pyrexias not influenced by malaria. In the pyrexia of typhus, too, there is commonly an evening exacerbation, during which the temperature may be from two to three degrees, or more, higher than that of the morning. In scarlatina, measles, variola, varicella, erysipelas, and in yellow fever, there usually are more or less regularly marked exacerbations of the fever during the pyrectic period of these diseases. In cases of chronic farcy, in the human subject, it is noted by Professor

Arthur Gamgee, that the disease "is accompanied by fever which often assumes a tertian type" (Reynolds' *Medicine*, vol. i, p. 327).

In pyæmia, too, Dr. Bristowe observes (*Medicine*, 2nd edit., 1878, p. 270) that the rigors sometimes assume a quotidian character, and that "the temperature of pyæmia has a good deal of resemblance to that of ague". Every surgeon will have noticed how frequent in occurrence are the evening exacerbations of fever in phthisis. In the symptomatic fevers accompanying several inflammatory diseases, such as acute rheumatism, and even pneumonia, the same fact has been observed. We are, then, justified in concluding that mere periodical exacerbations and remissions may, with more truth, be said to be phenomena accompanying almost all pyrectic conditions of the body, than characteristic of pyrexias of malarial origin. In what sense then, you may ask, is "periodicity" to be understood as especially characteristic or diagnostic of fevers of malarial origin? The answer is that in the one group of fevers easy of diagnosis, and indisputably of malarial origin—I mean the intermittents—the pyrectic periods are characterised by an almost certain regularity in their periodical recurrence; indeed, the most certain and most characteristic symptom of these fevers is the recurrence and subsidence of the pyrexia at intervals so regular and invariable as to admit of these fevers being classified into well-defined types—quotidian, tertian, quartan—and even into such secondary types as "double tertian", "double quartan", in which the periods of recurrence of the disease are, though complex, still almost invariable.

In the fevers classed as remittents this character of periodicity is not so marked, indeed is often scarcely more so than in the many fevers which are admittedly not of malarial origin. The question as to the true nature of many of the fevers at present classed as "remittents" can scarcely be considered as settled. Probably, many of them are in reality cases of enteric fever, or typhus, and being, therefore, of non-malarial origin cannot be expected to bear the marked characters of malarial fevers. The most characteristic symptom, then, of those fevers which are certainly known to be of malarial origin is their almost invariable periodical recurrence, or exacerbation, after certain definite intervals of time; and in this precise sense alone is the term "periodicity" characteristic of malarial diseases.

Another well-known fact concerning malaria is that this poison not only causes its fevers to assume this periodicity, but, in a person impregnated with the poison, stamps a simi-

lar impress of periodicity on any affection from which he may suffer while living in malarious regions. Even such a disease as pneumonia not infrequently assumes a distinctly periodic form in its pyrexia, the exacerbations in some cases occurring daily; in other cases on each second or third day, which latter has, I believe, never been observed to occur in non-malarious regions.

I have, on more than one occasion, seen distinct pneumonia returned as ague on account of this impress of periodicity. The fever of acute rheumatism frequently shows this impress. In one case under my care it took the tertian ague form. In another case, the pains of dysmenorrhæa took the form of periodicity of a quotidian ague, returned at the same time, and lasted the same number of hours each day. This peculiar impress of periodicity may be acquired, although the patient may never have suffered from distinct fever of the type assumed in these various outstanding complaints; it seems to be acquired in the same slow, insidious, way that the malarial cachexia is.

Among persons who, after having spent many years in India, have retired to England, where the climate has, of late years, been practically free from malaria, it is far from uncommon to find that neuralgic pains, and pains in the cicatrices of old wounds have a distinctly periodic character, return and leave at certain hours, or on certain days, and retain this peculiarity during life. Any disease, too, which such persons may acquire in England will tend to develop a tendency to periodical recurrence in some of its symptoms, most frequently in its pyrexia.

I should like to enlarge on a point previously alluded to in a cursory way, viz., the extreme difficulty frequently introduced into the diagnosis of enteric from remittent fever in India, owing to this marked impress of periodicity cast over the pyrexia and symptoms of enteric by malaria. As previously noted, the pyrexia of non-malarious typhoid often so closely resembles that of true malarial remittent that the diagnosis of the one disease from the other is in no way assisted by the thermometric indications.

On the resemblance between malarial remittent and enteric fever, I have already quoted the authority of De Claubry, Trousseau, and John Harley. I may further quote from Murchison (*Continued Fevers*, 2nd edition, p. 592.) "The diagnosis between enteric and remittent fever is frequently extremely difficult in countries where both prevail together. The pyrexia of enteric fever is essentially remittent, and cases have occurred in my own practice, and been noted by

Trousseau and other observers, especially in malarious countries, in which it has put on at first an intermittent type. Moreover, vomiting and diarrhœa may occur in both diseases, while enlargement of the spleen, cerebral symptoms, and the typhoid state are common to both. The eruption is, perhaps, the only distinctive mark of enteric fever to be relied on, and, in every case of remittent fever, complicated with abdominal symptoms, it ought to be carefully looked for. The close resemblance of enteric to remittent fever accounts for the fact that it is only within the last few years that the former malady has been recognised as occurring in India." Authorities on this subject abound in medical literature.

With regard to this all-important point of clear diagnosis between enteric and malarial remittent, we shall probably find that a careful comparison of the whole train of symptoms of the two diseases, rather than the laying main stress on any one symptom, will best serve to lead to a diagnosis, in spite of the additional mask of periodicity with which malaria may have still further exaggerated the normal remittent tendency of an enteric fever.

I shall endeavour to give a brief analysis, by comparison and contrast, of the main symptoms of these two diseases, with a view of illustrating some of the chief differences in their resemblances. We may consider the symptoms in the following order: 1, the pyrexia; 2, rash; 3, the abdominal symptoms; 4, the cerebral symptoms; 5, therapeutic test of the action of quinine.

1. *The Pyrexia.*—Much has already been said, and several authorities have been quoted, in the earlier portion of this paper, to illustrate the remarkable resemblance between the pyrexias of enteric and remittent fevers.

Bristowe also notes (*Medicine*, 2nd edition, 1878, p. 286) "Remittent fever presents, at first sight, an almost closer relationship, in the type of its fever, with enteric . . . . . than with the varieties of intermittent fever." Either disease may come on slowly, or be sudden in its invasion. But, as noted by Bristowe (*Medicine*, p. 218) it is only in exceptional cases that typhoid is sudden in its invasion. On the other hand, it is far from uncommon to find malarial remittent come on suddenly and rapidly after exposure to malarial poison in the intense form in which it exists in terrais and swampy jungle in the unhealthy seasons. In both forms of fever, the exacerbations usually occur at night, from evening to midnight; while, in cases of enteric occurring in malarious regions, exacerbations may occur twice in the twenty-four hours, and at periods as regular as in a true remittent. On nearly every

other point the pyrexias of the two diseases may, and often do, present identical characters.

2. *Rash : its Presence or Absence.*—Murchison notes that the eruption in enteric fever is, perhaps, the only distinctive mark to be relied on as diagnostic. The main characters of this rash are : that it occurs in the form of successive crops of rose-coloured spot, elevated, disappearing on pressure, each crop lasting about three days, appearing chiefly on the abdomen, chest and back, occasionally also on face and extremities.

In Europe, the rash is entirely absent in about one-fourth of the cases of typhoid, and even when it is present not more than a dozen spots may be discoverable. Incomplete as this symptom is, then, in Europe, it becomes, in India, a very broken reed to lean upon for assistance in diagnosis. For, in the case of the native races of India, such a rash could rarely be distinguished on skins whose colour varies from a copper, or coffee-tint up to jet black. Moreover, the paler skin of the European tends, as a result of over-stimulation by the heat of India, to show crops of spots—of Lichen Tropicus (prickly heat), of Strophulus, and other rashes—even at ordinary times; while, during a fever, the skin, owing to excessive action, is almost sure to show crops of such spots, and is often covered with miliaria and sudamina. The spots of these innocent rashes, like the typhoid eruption, also tend to develop themselves on the abdomen, chest, and back, rather than on the extremities ; and, during a fever, they are not uncommonly present in such densely aggregated masses as to destroy all possibility of differentiating from among their overwhelming multitudes any spots which may be present specifically due to typhoid.

There are, therefore, the following elements of uncertainty in the diagnostic value of the typhoid rash—25 per cent. of the cases are unaccompanied by rash—on dark-coloured skins the rash is not discoverable ; on pale-coloured skins in hot climates the presence of innocent rashes may obscure or swamp it. While, considering the condition of the skin, I may note that a jaundiced condition of it is far more frequently met with in malarial remittent than in typhoid.

3. *Abdominal Symptoms.*—These most frequently furnish, in India, the chief aids to diagnosis ; but even here many elements of uncertainty may exist. In typhoid, we expect to find abdominal pain, tenderness, and tumidity, with vomiting and diarrhœa. In both typhoid and malarial remittent there is usually some amount of tumidity of the whole abdomen from inflation of the gut, chiefly the colon, with flatus ;

and, consequently, some amount of pain on pressure over the whole region about the distended gut; yet the site of severe pain is widely different in the two diseases. In typhoid, the ileum immediately above the ileo-cæcal valve is the region in which the morbid process is always most extensive and advanced; consequently the right iliac fossa is the main region of severe pain on pressure, even gentle manipulation causing pain and gurgling; while the epigastric is not more painful on pressure than any other region of the tumid abdomen. In malarial remittent, the stomach and duodenum are the portions of the alimentary canal chiefly affected by the morbid process, and consequently the epigastric is *par excellence* the region of intense pain on pressure; and vomiting tends to be a most easily-excited, obstinate, and persistent symptom; while the right iliac region merely shares the modified tenderness of the tumid abdomen generally. In malarial remittent this uncontrollable tendency to vomiting is often one of the greatest difficulties in the way of treatment; the slightest pressure over the epigastrium, even of the patient's hand, or bedclothes, may at once induce it, and the administration of food or medicine by the mouth is often rendered impossible, as even the introduction of a few drops of iced water into the stomach is sufficient to set up the most obstinate and distressing vomiting. This is especially the case during the stage of exacerbation of the pyrexia. In my own practice I have been obliged, in several cases, to administer nourishment by the rectum, and quinine hypodermically.

The vomiting of simple typhoid seems rarely to approach, in persistency and severity, that common in malarial remittent. Few who have treated severe cases of remittent fever will forget the distress to the patient, and, often, anxiety to the physician, caused by this most obstinate symptom, vomiting, which has seemed determined to paralyse all efforts at treatment. The presence of blood in the vomit, which is consequently darkened or black, is much more common in malarial remittents than in typhoid, and the reason is apparent. The diarrhœa of typhoid is almost always present, and is more or less persistent throughout the existence of the disease. It consists of pale-yellow, pea-soup like, liquid stools, often containing shreddy matters from intestinal ulcers. These stools may, at advanced periods of the disease, contain discharges of blood; this blood may be either bright red, or dark in colour, is usually fluid, and may be very copious in amount, sometimes sufficiently so to cause the death of the patient.

In malarial remittent the characters of the bowel sym-

ptoms are usually in contrast to those obtaining in typhoid. Diarrhœa is probably a less common symptom than constipation. The use of aperients is frequently necessary in the early stages of remittent, and, in many cases, there is a marked tendency to constipation throughout the disease. Whether aperients have, or have not, been given, the stools tend to be of a dark bilious colour, often greenish, and sometimes resembling tar, from the presence of blood, which has been acted upon by the digestive secretions. Blood is, probably, less often present in the stools in remittent than in typhoid, and is rarely, or never, of a bright-red colour, but blackish; and the reason for its dark colour is apparent, as, instead of having flowed from an ulcer, or breach of surface, in the lower portion of the gut—ileum—as in typhoid, this blood in the stool of a remittent patient has been slowly exuded from the congested surface of the stomach or duodenum, which are the portions of the alimentary canal mainly affected in remittent; hence, this blood has been blackened owing to the action of digestive juices on its lengthened passage from the site of its exudation down to the rectum. Elements of embarrassing uncertainty, however, may and not uncommonly are, introduced into the indications afforded by the abdominal symptoms of the case in various ways. For example, if the sufferer be in a condition of malarial cachexia, from long-continued residence in malarious regions, the bowel symptoms may closely resemble those of typhoid. For one of the commonest symptoms of this cachectic condition is the tendency to suffer from diarrhœa of pale, ochre-coloured stools. This diarrhœa supervenes on the slightest provocation, often, indeed, is chronic, and consists of semi-digested food in the condition of bile-tinged chyme; but in this there is usually an absence of the shreddy matters, so easily found on washing a typhoid stool. Again, if, as will frequently be the case in India, a patient now suffering from malarial remittent fever has previously had dysentery, the new disease may be sufficient to cause some imperfectly-healed dysenteric cicatrices to give way; in which case the occurrence of shreddy tissue, and of bright-red blood in the stools, may throw doubt on the case, and cause these stools to closely resemble those typical of typhoid fever, especially if, as often occurs, the recurring dysentery be accompanied by pale, cachectic, diarrhœa.

4. *Cerebral Symptoms.*—In both enteric and remittent, confusion of intellect, drowsiness, coma, and low muttering delirium, may occur; and, in both diseases, the patient may sink into what is understood as the "typhoid" condition, with

dry, brown, tongue, sordes on gums, and utter prostration. In typhoid, however, the cerebral symptoms tend to be more gradual in their invasion; in the early stage, headache, giddiness, and restlessness are present, but the mind is usually clear, and there is rarely delirium, even at night; to these succeed, during the second week, dulness and apathy, then drowsiness and delirium; which last is more frequently of a low, muttering, subdued, restless kind, than maniacal and violent.

The cerebral symptoms in malarial remittent are not unfrequently sudden in their invasion. In severe cases, headache passes rapidly into delirium in the earliest stages of the disease; and this delirium is most frequently of a noisy, violent, maniacal kind. This may subside into mere headache and drowsiness during a remission, and again recur with the exacerbation in the early stage of the disease; later on, it may either merge into a more or less continued, subdued, form of delirium, or may disappear gradually in favourable cases. In the latter stages of an unfavourable case of remittent, the patient commonly passes into what is known as the "typhoid" condition—intense prostration—low, muttering delirium—with tongue dry, brown, and fissured—lips and gums covered with sordes—subsultus tendinum—coma—death.

It will be observed how intimately the whole train of symptoms of these two diseases may resemble one another, and to what an embarrassing extent this resemblance may be still further increased by any malarial taint on the patient.

5. *Therapeutic Test.*—Apart from the evidence afforded by the symptoms, there is one other distinction between remittent and typhoid fever which deserves notice, viz., the therapeutic test of the action of quinine on the disease. In a case of true typhoid fever, when not complicated by malaria, quinine probably exerts no influence, for good, on the disease. On the contrary, in a case of malarial remittent the persistent exhibition of quinine acts as an almost unfailing charm in controlling the disease in any stage; while its free and early administration may be relied on to rule the most stormy features of the worst attack.

This fact sufficiently indicates the desirability of clearly diagnosing the true nature of the fever during its early stages. For, if a malarial remittent be treated without quinine on the general principles applicable to a case of enteric fever, on account of the strong resemblance of some of its earlier symptoms to this latter disease, the result will probably be disastrous; while if, in spite of its resemblances to enteric fever, the true malarial nature of a remittent be distinctly

recognised, and free exhibition of quinine be made during its earlier stages, the disease will be kept well in hand, and led on to a favourable issue. On the other hand, the indiscriminate trust in quinine as a panacea in every form of fever occurring in India must necessarily tend to the disappointment of the practitioner, and to throw discredit on the powers of the drug. For, in a case of true enteric fever, closely simulating the symptoms of malarial remittent, or even actually bearing a malarial impress, we shall be doomed to disappointment if we expect quinine to have any influence further than that of neutralising and striking out any malarial complication which may be actually present, thus leaving untouched the bare enteric fever, to be recognised and treated in a manner suited to its true nature.

True enteric fever may begin in the form of an ordinary malarial intermittent. It has already been noted that, in a malarious region, some amount of distinctly intermittent fever, or ague, will accompany almost any exposure, injury, or marked departure from ordinary health. An excess at the table, a severe fall, a sprain, a bruise, or fracture of a bone, will commonly be so accompanied. Similarly, it has been noted that pneumonia and other diseases are often so ushered in; and I have seen cases of pneumonia returned and treated as simple ague. It will, therefore, cause no surprise to find such a disease as enteric ushered in with those symptoms of intermittent fever which have been noted as tending, in a person exposed to the malarial poison, to accompany all deviations from health. But we are scarcely justified in overlooking the presence of the fractured bone, the pneumonia, or the true enteric fever, on account of the ague associated with these; nor should we deserve or attain success if we relied on quinine as a sufficient treatment of all or any of these conditions.

Having, then, penetrated beneath the thin veil of intermittent fever which may have ushered in either a remittent, or an enteric fever, we have to decide the frequently knotty question as to which type the case really and actually belongs, as in the one case quinine will act as a charm, in the other prove itself practically useless for good. Moreover, in malarious regions, the all-pervading poison of malaria may not merely cause some intermittent fever to usher in other diseases and forms of fever, and cast a thin veil of periodicity over their pyrexias, but it may be a sufficiently active and intense a poison to cause its own full train of symptoms, viz., true malarial fever, intermittent, or remittent, to complicate and accompany certain other diseases throughout their whole course.

I have watched and treated cases of pneumonia, and of acute rheumatism, in which fever of a tertian type has accompanied the disease all through. Fever of a quotidian type is less clearly recognisable as certainly due to malaria, as much must be allowed for the normal tendency to evening exacerbations of the pyrexias of most diseases. When a distinctly malarial complication accompanies, and is bound up with true enteric fever, we have the disease that has been recognised, and described by numerous writers of divers periods and lands, as paludo-enteric, or typho-malarial, fever. Not that the malarial poison, and the typhoid poison can be produced, either of them indifferently, from general causes; nor that the poison of malaria can produce typhoid, or the poison of typhoid give rise to malarial fevers, but that the two poisons have both been introduced into the same system, and have happened, both of them, to fall on conditions favourable to the growth of each; and, consequently, each has produced its own effect, each has fulfilled its own law of development. Just so, we sometimes see the sober colour of a field of corn rendered bright by scarlet poppy flowers, as a result of presence of poppy seeds among the corn scattered by the sower, and of the ground having been suited to the development of each. But who, on this account, would look to corn seeds to produce him poppy flowers, or poppy seeds to grow him corn, or to either one of these seeds to germinate indifferently into poppy and corn plants?

Typho-malarial fever may be taken as affording an illustration of the parallelogram of forces; the malarial poison, and the typhoid poison, being the two forces acting on a point—the patient—and being represented in magnitude and direction by the two sides of the parallelogram; each produces its full effect in its own proper direction; the resultant being represented in magnitude and direction by the diagonal through this point, *i. e.*, by typho-malarial fever, a type not taking precisely the normal direction of a typhoid case, nor of a malarial fever case, but distinctly and clearly of the nature of both conjoined and interacting. I believe that this disease, typho-malarial fever, has only been recognised in regions combining the conditions favourable to the development of both malarial poison, and of the typhoid poison.

Regions known as malarious are most frequently low-lying, with respect to their surroundings, of very imperfect natural drainage, and capable of efficient artificial drainage only with difficulty, and at great expense. Hence, if a camp, town, or village, be situated in such a region, it is almost certain that impurities of fæcal as well as of vegetable origin

will abound; and there will be present every condition favourable to the development and spread of both the typhoid poison and of the malarial poison.

A long array of authorities could be quoted on the subject of the prevalence of fevers of a mixed malarial and typhoid type. First, Lancisi, writing of the Roman marshes, lays great stress on the occurrence of epidemics of pernicious malarial fever which, beginning as simple ague, usually tertian, always intermittent at its commencement, turned into continued fever. He further describes some of these cases as showing, among other conditions diverse from intermittents, such symptoms as parched tongue, delirium, feeble voice, livid spots on the skin, swollen, tense, painful abdomen, frequent dejecta of a pale colour, often containing liquid blood, ending with coma, cold sweats, and discharge of pale urine.

I may give Lancisi's own forcible words. In *Lancisii Opera*, liber ii, "De Noxiis Paludum Effluviis", caput v, p. 158-9, in the chapter entitled, "Duplex febrium epidemicarum natura, et quæ maximæ ægros symptomata afflixerunt", he says, in describing these cases, "..... Ceterum lingua erat arida et suboscura; verum accedente mentis motione, nulla ut plurimum querula sitis audiebatur .....; papulæ in cute lividæ; .... venter elatus, tensus, ante delirium dolens, ac frequenter post sextam pallido-biliosis ac fætidissimus, non raro etiam cruentis liquaminibus solutus." A very good description of the symptoms of enteric fever.

He further describes the *post-mortem* appearances of these cases in liber ii, Epidem. I, cap. vi, p. 161, "Intestina autem fere undique sphacelo-tentata continebant fœtidissima excrementa, vermiumque copiam, imo veró circulares hic illic subnigras maculas exhibebant, quarum in centris occisuras quasdam insculptas vidimus." Cases of dysentery seem included with cases of enteric fever in this description.

See also *Idem.*, lib. ii, Epidem. v, cap. ii, p. 342. I think you will agree with me that many of the cases, whose symptoms and *post-mortem* appearances are so well described by Lancisi, would now be set down as cases of typho-malarial fever.

Again, Mr. Chas. Mayo reported that the "camp fever" of the army of the Potomac was generally recognised as a typho-malarial fever, in which the symptoms of typhoid fever, diarrhœa, rose rash, etc., were associated with those of intermittent fever. The typhoid symptoms occasionally predominated, and *post-mortem* examination revealed lesions of Peyer's patches.

The Walcheren fever seems to have been of the same type.

Dr. Davis describes this Walcheren fever as assuming the quotidian, tertian, double tertian, or even remitting type, often changing to the continued character. In several of his cases there were *post-mortem* evidences of lesions of the agminated and solitary glands of the small intestine.

Professor J. Harley considers that many of Dr. Davis's cases were of the typho-malarious character. Professor J. Harley erects paludo-enteric fever into a distinct variety of enteric fever, and records his belief that this is the most common form of the disease—typhoid—and that the evidence of the direct connection between the two diseases, ague and typhoid, is irresistible.

More recently, new evidence has been adduced as to the co-existence of malarial with typhoid fever. Thus, I find in the *Practitioner*, December 1880, p. 474, a quotation from Sir Joseph Fayrer's address to this (Epidemiological) Society on November 3rd, 1880, viz.: "Typhoid was the principal disease which affected the troops during the campaign in Zululand, and Mr. Ross says the disease affecting the troops shut up in Ekowe was called typho-malarial fever, with rose-coloured spots and affections of Peyer's glands."

Again, I find, in the *Dublin Journal of Medical Science*, January 1881, p. 17, a very able and interesting paper on "Enteric Fever, and its Modifications by Malaria, in South Africa", by Surgeon-Major R. W. Jackson, C.B., A.M.D.

Professor J. Harley, moreover, notes that of sixty-eight cases of enteric fever which came under his care in the autumn of 1865, the bulk came from "the low-lying districts on the banks of the Thames within and about the Metropolis, where ague was formerly so rife, and from certain of the filthiest and most crowded parishes; while only five cases came from the more elevated localities."

The conclusion I wish to draw from these quotations from various authorities concerning typho-malarial fever is this:—That there seems a general consensus of opinion that the conditions which favour the development of malaria, also are favourable to the development and spread of typhoid fever, viz., want of drainage, non-removal of nuisances, and a general unsanitary condition, all of which tend to occur in low-lying or swampy regions.

Thus, in a report, by Dr. G. Whitley, as to the amount of ague now prevalent in England (*Sixth Report, Privy Council*, 1863), on pp. 432, 451, 452, several instances are recorded of places noted for prevalence of ague being also noted for the prevalence of typhoid.

It must be conceded that in such very diverse regions as

the Pontine marshes of Rome, at Walcheren, on the Potomac, in the at-one-time ague districts of England, in Zululand, and South-East Africa, there has been gradually accumulated a mass of evidence that the conditions producing malaria are also favourable to the production of typhoid, and that these two diseases, in consequence, tend to go hand in hand, or co-exist in an epidemic form. How, then, are we to escape the conclusion that, in India, the same conditions and factors must produce the same results?

Probably, no regions of the known world are more a prey to malaria than the plains of India. Is it not surprising to find that so few cases of typhoid have, until late years, been recognised and described? Is India an exception to the natural laws which long-accumulated experience has shown to hold in almost every other malarial region of the globe? Are we to hope that the conditions known to favour the production of typhoid in other countries, and which abound in India, cease to be operative in producing it in that country—that cause and effect are there divorced?

Arguing from analogy, we should, on the face of it, be led to expect that, in a country in which the bulk of admissions to hospital are for affections due to malaria, cases of typhoid should be recognised as numerous, and that the prevalence of this disease should have been, from early times, placed beyond doubt or question.

Putting aside, however, deductions from analogy, as being of merely a relative value in such an exact science as we desire medicine to become, we may adduce the evidence of facts, observed and recorded by physicians in India, as showing that typhoid—probably, in many cases, of a typho-malarious nature—is by no means uncommon there, and that its production and spread are governed by the same laws, and influenced by the same conditions, in India, as in those other and diverse parts of the world, in which malarious regions have been recorded as notoriously furnishing conditions favourable to the production of typhoid.

At one time it was generally held that true typhoid did not occur in India; to this pre-typhoid era there succeeded a cycle of years during which some, though not numerous, cases were recognised and recorded; while, even at present, it is, perhaps, a not universally admitted fact that typhoid is prevalent there, as in other countries which abound in the conditions known to contribute to its existence and spread.

Probably, in no other country are there so many difficulties in the way of a clear differentiation of typhoid as in India.

The frequency of that disease being ushered in by, overcast by, or throughout its whole course complicated with, malarial fevers—the intimate resemblance its symptoms often bear to those of remittent—the extreme prevalence in India of diarrhœas, chronic and acute, often pale-coloured, and containing blood when dysenteric, and the frequency of these diarrhœas being associated with remittent and other malarial fevers; all these causes have militated against a clear and general recognition of typhoid fever, and will, perhaps, always furnish arguments for denying its existence or prevalence.

Probably, future study of the class of fevers, grouped as remittent, will show many of them to be of the nature of typhoid —some of typhus.

While on this subject of the too frequent obscurity and insufficiency of the diagnostic indications of fevers in India, I would ask your indulgence to permit me to recount an anecdote told by one of the classical satirists of a certain figure-painter whose pictures were so execrably drawn that it was almost impossible, from a study of them, to fathom the design of the artist; hence, it was found necessary to assist the spectator's imagination by writing clearly beneath each painting, "This is a cock." "This is a bull."

If I may be excused the levity of the application, I would say that in certain forms of our Indian fevers the design and intention of nature, as to the type of fever aimed at (so to speak), is, in many cases, so obscured by a host of points of similarity to, and divergence from, a plurality of types, that it needs a very close study and digest of these cases to enable us to confidently proclaim, "This is remittent;" "This is typhoid;" and we cannot, unhappily, in this case, as in the former, compel the artificer—Nature—to herself label her own indifferently indicated design.

THE NATURE AND ORIGIN OF CLIMATIC FEVER.

By Surgeon-Major CHAS. F. OLDHAM, M.D.

[*Read: January 5th*, 1881.]

In this paper I have endeavoured to point out, as clearly and concisely as may be, the nature and origin of climatic fevers. It is undeniable that a great diversity of opinion exists on this subject; and it is evident that, until the nature of a disease be defined, all preventive measures directed against it must be merely empirical—mere groping in the dark.

Climatic fevers, which are now, happily, little known in England, but which still cause a very great mortality in other countries, may be divided into two great groups, viz.:

1. Those which are caused by excess of heat.
2. Those which arise from a deficiency of heat, or, in other words, from chill.

Of the first of these groups, insolation and the ardent fevers of hot climates are examples. These are continued fevers, but they are intimately connected with fevers of the periodic type. They are usually confined to tropical climates; but they sometimes occur during very hot seasons in countries without the tropics; and this is especially the case when the high temperature sets in suddenly.

These fevers, it is generally allowed, are caused by the direct action of great heat, and not by any specific poison. They, in fact, consist essentially in the accumulation of heat in the system, as a consequence of the failure or embarrassment of the functions concerned in its equalisation, or rather in its diminution.

As might be expected, therefore, the liability to these fevers is very greatly increased by any circumstance tending to interfere with the functions referred to. Thus, soldiers, when marching in hot weather, are very liable to attacks of insolation, unless frequent halts are allowed, and they are supplied freely with water.

The second great group of climatic fevers includes all the periodic or so-called malarious fevers. These disorders are

very widely distributed, and they have always been so prevalent in those eastern countries which were the nursery of medical science, that a large portion of the writings of the ancient physicians is devoted to them. Opinions as to the cause of these fevers have varied much. At different times they have been ascribed to—

1, exhalations from decomposing organic, and especially vegetable, matter in water or in soil; 2, the drinking of water impregnated with similar matters; 3, electrical action; 4, the influence of certain geological formations; 5, the action of certain gases developed in swamps; 6, the invasion of the body by certain low forms of organic life; 7, climatic influences.

The last of these ascribed causes should perhaps have been mentioned first, as there can be no doubt that the ancient physicians believed in the climatic origin of these diseases. This belief is held to this day by the native medical practitioners throughout the east.

Of the old writers, Avicenna appears to have been the first to ascribe noxious effects to the exhalations from water containing decomposing vegetable matters, such as flax. But this authority, who has been followed blindly by successive writers down to the present day, attributes the same properties to water impregnated with bitumen. It is evident, therefore, that he must have been misled, as others have been, by the action of the fœtid smell upon his imagination.

That climatic fevers frequently prevail in marshy places is certain. In temperate regions, they rarely occur in other localities. The reason for this I will explain hereafter. The constant connection of these fevers with marshes, in the countries with which the writers on the subject were best acquainted, led to the idea that the cause must be a paludal poison generated only in swampy places. Moisture generally favours the growth of a luxuriant vegetation which, from its associations, came to be looked upon as the source of the poison. To this day, even, rank vegetation is attacked by energetic sanitarians, under the vain idea that in destroying it they are removing the noxious properties of the swamp.

Admitting fully the preference of the periodic fevers for marshy localities, it must be observed that some such places are free from them, or nearly so, although abounding in vegetation as luxuriant, and as liable to decay, as that of the most deadly swamps.

This fact alone should be fatal to the theory which ascribes "malaria" to decomposing vegetation. But, when we find

the diseases ascribed to this "paludal poison" occurring in their deadliest forms where there is no vegetation to decay, the proof that vegetable decomposition is not the cause of climatic fevers is positive, and further argument useless.

Time will not admit of my quoting many instances, in support of what I have said, but more may be found in my work on malaria.* I may, however, refer to the bogs of Ireland, which have so long been a puzzle to writers on paludal poison.

There can be no question that the bogs consist, for the most part, of more or less decomposed vegetable matter, and that they present all the most prominent features of swamps. The people of Ireland, however, suffer little from "malarious" fevers.

Some writers have, indeed, affirmed that these disorders are unknown in Ireland, but this is not strictly correct. There have been endeavours to explain the comparative immunity of Ireland from "paludal poison" by supposing that the bog-water possesses certain antiseptic properties. But, whatever these properties may be, they do not prevent vegetable decomposition, as the "bog-holes", or pools, are used in many places for the purpose of rotting flax. Moreover, the steeping of flax, upon which so much stress has been laid by all advocates of the decayed vegetable theory, is admitted to be, in Ireland, absolutely innocuous, whether bog-water be used or not.

One other instance—this time a tropical one—Mattang, the port of Larut, which was the base of operations for the Perak expedition, in 1875-6, is built in a mangrove swamp near the mouth of the Larut river. The site is barely above the sea-level at high tide; and, to consolidate the ground sufficiently for roads or for buildings, the mud is dug up and heaped upon the spot to be raised. This mud has a disagreeable marshy smell, and the excavations left are filled with stagnant water. The vegetation is rank; and the heat, I need scarcely say, is great, as the place is some five degrees north of the equator.

If stagnant water and decomposing vegetable matter generate the marsh poison, Mattang should be a pest-house; but it is nothing of the kind. The resident magistrate, and other officials, assured me that the place was remarkably free from intermittent or remittent fevers, and the troops passing through it scarcely suffered at all from these affections. Of course the objections which apply to the theory that supposes the cause of climatic fevers to be putrescent matters

* *What is Malaria?* etc. London: H. K. Lewis.

in water, apply equally to the supposition that it arises from decomposing matters in the soil.

The idea that fevers and diseases of the spleen are caused by drinking marsh-water is very ancient, and its origin may easily be traced to the fact that habitual drinkers of marsh-water must live in marshy districts.

An outbreak of fever, which occurred many years ago on board the French transport *Argo*, mentioned by M. Boudin, is often quoted as a proof that intermittents may be caused by drinking impure water. But Boudin himself did not consider that it proved anything of the kind. He expressly says: "Au premier bord les faits qui précèdent paraissent décisifs, et il semble impossible d'attribuer les fièvres paludéennes de *l'Argo* à une cause autre que l'usage d'une mauvaise eau. Cependant, on voit que les passagers militaires de *l'Argo* quittaient un foyer marécageux, dans lequel ils avaient séjourné plus ou moins longtemps, et dans lequel ils avaient puisé au moins une prédisposition aux fièvres paludéennes."*

That marsh-water cannot, in any shape, be the cause of climatic fevers becomes evident, if we reflect that these disorders are not confined to swamps, or to those who have drunk the water of such places, but prevail in the most arid and barren districts. They are very prevalent in the great desert of Northern India, where the water is obtained from wells of enormous depth, and cannot possibly be contaminated by vegetable decomposition; and they occur at Aden, where distilled water is used, or rain water stored in cisterns excavated in the rock.

The electrical state of the atmosphere, to which climatic fevers have by some writers been attributed, cannot be the same under all the varied conditions in which these disorders prevail. These fevers are confined to no climate, to no season, to no altitude, and to no especial meteorological conditions. They are common in countries and in seasons in which electrical disturbances are frequent and violent; and in others, where thunder and lightning are almost unknown.

The geological formation of countries in which climatic fevers prevail has naturally attracted the attention of many observers, some of whom have considered that the origin of these disorders may be traced to particular rocks or soils. But, on comparing the results of investigations made in different regions, it is evident that there is scarcely any description of soil or rock in connection with which they do not occur; and that formations for which, in some districts, they

* *Traité de Geographie*, etc., ii, 142.

appear to show a preference, in others are almost entirely free from them.

The great prevalence of climatic fevers at Hong Kong in 1864, is often quoted as a proof of the origin of these disorders in some poison given off by the soil or rocks, when exposed by excavation. The example, however, is an unfortunate one, for several reasons, viz.: 1. The same type of fever prevailed at Hong Kong before the excavations commenced. 2. The men were exposed (to an extent unheard of in India) to climatic vicissitudes which would have been certain to cause fever in any tropical climate. And this excessive exposure was contemporaneous with the excavations to which the fevers were ascribed. 3. When the exposure of the men was diminished the outbreak subsided. 4. Those men, whose duties did not expose them to the sun by day, and the chill damp air and dew at night, escaped fever, although they lived as near to the excavations as their comrades.

To all this may be added that, when specimens of the rock and soil were submitted to analysis to discover the nature of the poison, they were found to contain no noxious ingredient, and less than two per cent. of organic matter. It would have been more to the purpose to have analysed the roofs of the huts in which the men were quartered, as a reference to the *Blue Book* will show.

That climatic fevers do not arise from sulphuretted or carburetted hydrogen, or any other gas developed in swampy places, is now so generally admitted that I need not further allude to them; so I will pass on to the germ or parasite theory. Several writers have endeavoured to trace the origin of climatic fevers to the invasion of the body by low forms of organic life.

So long ago as 1866, Dr. Salisbury announced the discovery of the cause of malarious fevers in the spores of some low forms of vegetation existing in swamps. This writer reported that he had found these vegetations abundant in marshes, and on the surface of newly-excavated earth in swampy places; also that he had found the spores in the sputa and urine of patients suffering from intermittents.

Dr. Salisbury considered also that "the tendency to periodicity in all febrile diseases probably marks the periods of the successive crops of vegetation".

Later writers, British and foreign, have adopted similar views, and have reported further discoveries in the same field. So far as climatic fevers are concerned, however, none of these discoveries have been confirmed. Nor are they

likely to be so. Even the *Bacillus malariæ* is no more the cause of intermittent fevers than any of the organisms which preceded it, and were, each in turn, announced as undoubted ague-germs.

All the low organic forms which have been supposed to give rise to periodic fevers are of paludal origin and habit; a moist atmosphere and a damp soil are essential to their existence. What, then, can be the cause of the same diseases, where marshes do not exist, in dry and parched countries, where, amidst sandy deserts or barren rocks, these fevers prevail in their severest forms?

That germs and parasites exist I do not doubt; but they do not cause climatic fevers. It is easy to understand the possibility of a contagious disease having its origin in some form of living organism; for we may imagine the germs, on becoming ripe, making their escape from the body of the patient. In these cases we know that the surrounding atmosphere becomes contaminated, and that persons exposed to it are liable to be attacked by the same disease; the clothing, too, of the sufferer becomes infected.

But, if climatic fevers be caused by similar organisms, what becomes of the successive crops or broods produced during a case of ague? They must leave the patient, or he could not recover; they do not infect the atmosphere or the clothing, as the disease does not spread through them; they do not invade the bodies of the attendants, or these would be attacked by the same disease.

If, after reproducing itself within the human body to such an extent as to cause the symptoms ascribed to it, the whole living mass—plant and germ, parent and offspring—suddenly ceases to exist, what puts an end to its life, and what becomes of the debris?

Again, if after passing through animal bodies these organisms are incapable of reproducing themselves, they must exist independently in every locality in which climatic fevers prevail. They must, therefore, in hot climates live and thrive, not only in marshes and swamps, but in dry sandy deserts, and amongst arid rocks. They must be in their fullest vigour when the earth and air are so perfectly dry and parched that all low forms of organic life are desiccated and burned up; and also when both soil and atmosphere are saturated with moisture, and drenched and scoured by the tropical rains. Yet, in cooler climates, these same organisms must be unable to exist, except in the vicinity of marshes.

However applicable the germ theory may be to those diseases in which a poison is developed, capable of reproducing

itself, and of being communicated from man to man, it is impossible that climatic fevers which are not infectious can arise from the same cause.

Since 1870, when I first endeavoured to show how untenable were the theories held as to the nature of malaria, a considerable change seems to have taken place in the opinions held on the subject. We now hear little of the poison derived from decomposing vegetable matter, which was then the orthodox belief. And, although many still cling fondly to the idea of a specific poison, no definite theory seems to prevail as to its nature and origin.

When brought face to face, in India, with the various forms of so-called "malarious" disease, I found it impossible to account for what I saw under any of the then-prevailing theories. After a careful study of the subject, extending over several years, I arrived at the conclusion that malaria, as a specific poison, did not exist. Further, I became convinced that the diseases usually attributed to malarious influence were caused by chill, or, in other words, by the sudden abstraction of animal heat. I found also that the greater prevalence of "malaria" in hot climates, and the graver type of disease produced there, could in this way be readily accounted for. From this point of view, moreover, the intimate connection of so-called "malarious" fevers with pneumonia, rheumatism, dysentery, hepatitis, and other climatic diseases, hitherto so incomprehensible, became perfectly clear and distinct.

I must apologise for observing here that, in using the word "chill", I meant to express the undue abstraction of heat from the human body—not the cooling of a thermometer or of the atmosphere—a distinction which seems to have been lost sight of by some of those who have discussed my views. The subject is not, therefore, a mere question of meteorology; and variations in the temperature of the air have less influence than the degree of exposure to them; while more important perhaps than either is the condition of the heat-generating powers of the system.

To this condition of the heat-generating powers, I must beg your especial attention, as it forms an essential part of my views. It has been hitherto persistently ignored by most of my critics; and yet upon it hinges the whole question of the greater prevalence of climatic fevers in hot climates and hot seasons, as well as the graver type assumed in the tropics by this class of diseases.

It has long been recognised that, after continued exposure to great heat, the system becomes more sensitive to, and less capable of resisting, the effects of cold; and that, after a re-

sidence in the tropics, or even after a long period of hot weather in cooler regions, the body becomes sensitive to a fall of temperature which, in a milder climate or a cooler season, would scarcely be perceived.

Everyone knows, too, that unusual heat, when it sets in suddenly, is more distressing than when it comes upon us by degrees. In the same way, those who have been long inured to a hot climate suffer less from the high temperature than new arrivals. All this is because the heat-generating powers of the body are depressed by long-continued exposure to a high temperature, and are exalted by continuous cold. Owing to this compensatory process, which is, in fact, what is known as acclimatisation, the development of animal heat in the denizens of cold and temperate climates, is vigorous and active; so that a very great degree of cold can be borne without injury. Hence, to cause intermittent or remittent fevers in cool climates, cold requires to be supplemented by the powerful heat-abstracting influence of moisture. It is for this reason that, in Europe, agues are rarely met with, except in wet and marshy places. The chilling influence of the raw, damp, night air of swamps and marshes is well known, and is often described as "aguish". It is well known, too, that those who are exposed to it, without sufficient protection in the shape of clothing, shelter, or fire, frequently suffer from ague even in England. In hot climates, on the other hand, the heat-generating powers are so much less active that a very slight degree of chill, which would scarcely be noticed in cooler countries, may produce the most serious results. There, the slight reduction of temperature caused by a shower of rain, or a few moments exposure to the draught of air from a punkah or tattie, when the clothing is moist from perspiration, will often cause an attack of climatic fever, or some of its allied disorders, without any exposure to night air or dew. A similar condition is produced, but in a much milder degree, by prolonged hot seasons in temperate climates.

The acclimatising process, to which I have just alluded, and to which I attach so much importance, requires time to produce its effects. A transient exposure to heat or cold does not cause the same results, or does so in a very slight degree. Hence, stokers, glass-blowers, and others, who, in cool climates, are temporarily exposed to great heat, do not experience that depression of the heat-generating powers, which is produced by a prolonged residence in a hot climate. This also explains the benefit derived, by residents in the tropics, from occasional temporary removals to a cooler climate.

Let us now see how far these views agree with the circumstances under which climatic fevers are known to occur. That these fevers prevail in damp and marshy places is unquestionable. But they are not confined to those swamps in which vegetation is luxuriant, or in which water is stagnant or putrid. On the contrary, they often rage, as so graphically described by Fergusson, in damp and ill-drained places where vegetation, and even surface-water, are absent.

What peculiarity, then, is common to these two classes of swamp? Is there anything but the damp, and the chill, foggy, night air? This, too, they have in common with other haunts of "paludal poison", such as the moist alluvial delta, over-irrigated fields, or the dry bed of a river or torrent, which is often as deadly as the most poisonous marsh, though nothing but sand, shingle, or boulders may be visible. The forest, or jungle, when wet has the same characteristics as the marsh; when dry, it is no more unhealthy than other places.

Marshy localities, however, are not dangerous in proportion to the degree of moisture. If they were so, some of our most valuable Eastern possessions would be uninhabitable. Fevers, too, are not always most prevalent at the seasons in which humidity is greatest; as, except in equatorial countries, they generally prevail most when the "rains" have ceased, and the soil is drying up. Moreover, as I have already mentioned, the neighbourhood of swamps is not always unhealthy, and is sometimes singularly free from climatic fevers. In such cases it will be found that the climate is moist and extremely equable. The night air is harmless where it is not chilly.

Ireland is remarkable for the moisture and equability of her climate, and is famous for her bogs and marshes, yet climatic fevers are rare.

Singapore and Malacca, as well as Mattang, which I have already mentioned, are hot, moist, and surrounded by marshes, with a profuse tropical vegetation. Yet these are not hotbeds of paludal poison, but are noted for their exemption from climatic fevers. In each case the equability of the climate is remarkable. This is partly due to the constant humidity of the atmosphere, which checks the nightly radiation of heat. Hence, it is possible in a moist climate to live on the borders of swamps, which, with a drier atmosphere, would be pestilential. Hence, too, the comparative absence of paludal poison in the rainy season of drier climates; and its intensity when the rains have ceased, and the swamps are drying up.

In the expedition to the Malay Peninsula in 1875-6, the Malacca column, of which I was principal medical officer,

marched for three weeks through swamps and jungle. The troops bivouacked amongst rank grass and reeds, or in the primeval forest, and this within some two degrees of the equator, yet scarcely a single case of fever occurred. The atmosphere was highly charged with moisture; the sky was cloudy; rain fell in frequent showers; and the climate was, at that season, so equable that but little difference was perceptible between the daily and nightly temperature. Damp, then, is not malaria, although so intimately associated with it. Something more is required to cause climatic fevers. And this is found in chill.

Where, as in Upper India, the year is divided into hot, rainy, and cold seasons, a very great degree of humidity in the rainy season does not cause unusual sickness; the climate then being mild and equable. But in autumn, when the rains have ceased, and both soil and atmosphere are drier, while sufficient dampness remains to intensify the effect of the chilly nights which then set in, climatic fevers become general.

To give an idea of the ravages caused by these autumnal fevers, I may mention that last year they attacked 75 per cent. of the population in the North-West Provinces of British India; and that the ratio of mortality, in the districts of Aligurh and Bulundshur, was 113 per 1,000. These figures were quoted, by the *Times* correspondent, from the *Official Gazette.*

Climatic fevers are in most countries very prevalent in autumn. Of course, the fall of the leaf and its decay have been supposed to account for this. But a still more striking peculiarity of this season than the fall of the leaf is the nightly cold, which is rendered the more trying by its succeeding rapidly to the continued heat of summer.

Here, I must remind you of what I have said of the predisposing influence of continuous heat. Climatic fevers are not, in all countries, most prevalent in autumn; but they are so everywhere at the period of transition from the hot to the cool season; and always more so, in proportion to the greatness and suddenness of the change. All this proves that the prevalence of these fevers in autumn is due to the climatic vicissitudes which then occur, and not to the falling leaves.

In equatorial regions where the climate is almost uniformly hot, and where, as Humboldt says, it only grows cold after storms of rain, the setting in of the monsoon or rainy season is the period at which the change from heat to cold is most marked; and it is at this season, and not in autumn, that intermittent and remittent fevers most prevail.

The marsh poison we are told is quiescent by day, when the sun is warm; but deadly at night, when the air becomes raw, damp, and chilly. Can there be any doubt, then, as to the nature of the poison? Can any poison appear in the swamp by night, which is not there by day, but the damp and chilly atmosphere? Moreover, this quiescence of the cause of climatic fevers, by day and its activity at night, is not confined to marshy places, but is to be observed wherever the temperature falls rapidly as the sun goes down. When the nights remain hot there is no "malaria", as already alluded to.

Climatic fevers often occur in their most deadly forms upon arid rocks, and in dry, sandy deserts which, unsheltered by trees or vegetation from the rays of a tropical sun, appear to have little in common with the marsh, except "paludal poison". But here, too, the temperature falls rapidly at night, and the heat by day is often so intense that the variation of temperature is very great. I will give an instance of this from my own experience.

In February 1872, at Bhawulpore, on the border of the great Indian Desert, the average daily maximum reading of a thermometer exposed to the sun was 107 deg. Fahr., while the average nightly minimim was 30 deg. Fahr. The daily average variation was 77 deg. Fahr. On twelve nights the mercury fell below 32 deg. Fahr., and on one occasion it fell below 20 deg. Fahr., the maximum for the same day being 107 deg. Fahr. The atmosphere was at this time so extremely dry that the nails cracked, and hair became almost brittle, no rain having fallen for four months. The difference between wet and dry bulb thermometers was from 10 deg. to 20 deg. The soil was parched, and almost all vegetation was dried up. Yet intermittent and remittent fevers, complicated with dysentery, pleurisy, and pneumonia, were general and very fatal.

One condition only is constant in, and common to, all the various situations in which climatic fevers of the periodic type extensively prevail; and that is a great and sudden fall of temperature. Necessarily, this fall of temperature usually occurs at night; and in most cases it is associated with a condensation of moisture, by which its effect upon animal bodies is intensified. This condensation of moisture is represented by the mists, fogs, and dew, so constantly associated with ague.

In all hot countries cold winds are dangerous, and the more so in proportion to the previous exposure to great heat. The present campaign in Afghanistan has shown this very

prominently. In the Cordillera of the Andes, and, in fact, wherever snowy mountains exist in a tropical climate, cold night winds are a fruitful cause of periodic fevers, as the natives of these countries well know. I once found a man lying rolled up in his blanket and shivering with ague on the Andrar Pass in the Himalayahs, at an altitude of 14,000 ft. On my asking him what was the matter, he said he had "eaten the snowy wind".

On board ships, in the tropical seas, periodic fevers constantly occur from exposure to wet and stormy weather; and this often after long periods of absence from any port.

In all ages, it has been known that the cause of periodic fevers is to be counteracted by fire; and that smoke has also a protective influence. To the present day this is recognised, both by the civilised man and the savage. Montfalcon tells us how, by means of large fires, Napoleon kept his troops in health in the marshes round Mantua. In Central and Western Africa, in Madagascar, Ceylon, Venezuela, and in India, except in the dry, hot, season, fires are kept up at night as a protection from fevers. In the Terai, or damp unhealthy forest, at the foot of the Himalayahs, the Goojur herdsmen and other tribes sleep round fires in the hottest weather, to keep off the dreaded "awąl". The effect of fire is to heat and dry the air, while smoke effectually checks the nightly radiation of heat. That fire and warmth could counteract any specific poison is impossible, but they are most efficient antidotes to chill.

The influence of shelter and clothing as protection from climatic fevers is generally admitted, even by those who ascribe those disorders to a specific poison; though how, or why, shelter or clothing should counteract any poison but chill, it is not easy to see.

The effects of insufficient protection from climatic vicissitudes were very evident in Cyprus. There, the troops were, in a semi-tropical climate, placed in single cloth bell-tents, which were intensely hot by day, and wet through by the dew at night. We may form some idea of the heat in these small thin tents, as we are told that the thermometer stood at 100 deg. Fahr., in a large hospital marquee. Yet some people could not account for Cyprus fever.

I cannot, within the limits of this paper, quote many cases in support of what I have said, but I may just mention my own personal experiences.

My first attack of intermittent was caused, in India, by sleeping with only a sheet over me between two open doors. The second was in the following year from sitting under a

punkah in an artificially cooled room, during a morning call, when my thin clothing was damp with perspiration from riding through the sun at midday. The third attack was seven years later, and came on in London, after exposure to a cold east wind. The fourth occurred on the borders of the Indian desert, and in the dry, hot, season. It was brought on by a cold bath. The fifth attack was caused, four years after, by a chill from passing a damp, shady, gorge in the hills, through which a cold wind was blowing down from the snow. The last attack, which was accompanied with bronchitis, occurred six years later, on the march to Kandahar in the present Afghan campaign. It was brought on by exposure to the cold piercing night wind, so well known to those who have served on the north-west frontier of India.

It is, I think, universally admitted that recurrent attacks of periodic fever may be caused by exposure to cold. It is not easy to see, therefore, why the primary attack should be supposed to require another origin. To be sure the recurrent attacks have been called relapses, and it has been argued that these may occur without any fresh supply of poison, or invasion of germs. But to talk of a relapse, after years of perfect health, seems to verge upon the ridiculous.

The same may, I think, be said of the long periods of incubation sometimes imagined to account for persons being attacked, for the first time, by climatic fevers who have not for months, nay, even years, been exposed to any paludal poison.

From the explanation which has been given of the origin of climatic fevers, it will be easy to understand the hitherto mysterious connection between these fevers and dysentery, hepatitis, pneumonia, rheumatism, and other disorders which are admittedly caused by climatic influences. Dysentery and hepatitis are so constantly associated with periodic fevers that they have been described as "malarious" diseases. Rheumatism is also a very common complication; but pneumonia is the most common of all, and is the cause of the greater part of the mortality from autumnal fevers in Northern India.

Time will not permit me to say much on the subject of paludal cachexia, although it is so intimately connected with climatic fevers. That a generally enfeebled state of health should prevail amongst residents in swampy places is not surprising. In cool climates this is almost invariably due to the effects of repeated attacks of ague. In the tropics, however, we have, in addition, the enervating effects of heat. Where the climate is very hot, moist, and equable, as it

usually is near the sea coast in tropical countries, the protean forms of disease known as paludal cachexia and masked malarial fever may prevail, even in the absence of intermittents. In such climates, periodic fevers are much less prevalent than where the variations of temperature are great; but their place seems to be taken by paludal cachexia, which is very common.

The effect upon the white race, of combined heat and moisture, is to produce anæmia; to destroy the tone of stomach and bowels; to relax, and enervate the whole system; and to cause a tendency to passive visceral engorgements and various neuroses—in short, all the symptoms of paludal cachexia. And, if we analyse the symptoms of so-called masked malarial fever, we find that they are such as would result, in a relaxed and atonic state of the system, from exposure to climatic vicissitudes too mild in degree to produce a sharp attack of intermittent or remittent fever.

The specific action of quina in cases of periodic fever, or paludal cachexia, is frequently quoted as positive proof of the origin of those diseases in a specific poison. And we constantly hear it said that such and such a complaint must have been caused by "malaria", as it yielded to quinine. For this assumption, however, there is no foundation—nay, more, quina has no specific action in these or any other cases. That quinine is very valuable in the treatment of climatic fevers I admit. That it is more so than any other drug with which we are acquainted, I do not dispute. But it is equally beneficial in a number of affections which are not in any way connected with any specific poison. It is not a certain cure for intermittent or remittent fevers; nor has it, beyond its powerful tonic effect, any influence in preventing their attacks. How, then, can it be a specific? In what does its specific action consist? I have known many instances of people being attacked by fever, who were in the habit of taking quinine daily. Dr. Livingstone observed that the fever was impartial, and attacked them whether they took quinine or not.

Surgeon-Major Davie, Principal Medical Officer of the Perak field force, in the Malay campaign of 1875-6, reports: "Malarious fevers did not show themselves till February, and, curiously enough, after the troops had been taking quinine regularly for some time." He adds: "It is probable that the quinine issue, although it did not act as a prophylactic against malarious fevers, was useful in improving the general health, and thus diminishing the liability to bowel complaints." And he says, further: "The Goorkhas got no

quinine as a prophylactic, and suffered in proportion very slightly more than 'the Buffs' from malarious fevers." As a fact, however, the Goorkhas suffered even less than the white troops, although they were more exposed, from being employed on escort duty. The men were singularly free from climatic fevers; as there was not one case of remittent, and but very few of intermittent. The officers took no quinine, yet not a single case of fever occurred amongst them.

Of the hundreds of thousands who every year suffer from climatic fevers in tropical countries, how large a proportion recover, yet how small a proportion of the cases is ever treated with quinine.

The long list of anti-periodic remedies of great repute, which are not supposed to have any specific action, shows that no such action is necessary to produce a curative effect. Moreover, quina is admitted to have no specific influence over those fevers of which a specific poison is known to be the cause.

In the months of August and September 1871, in the hospital of the Political Agent's Escort at Bhawulpore, twenty-seven sepoys suffering from intermittent fever were treated with quinine—part with three-grain doses three times a day, and part with doses of ten grains two hours before the paroxysm. At the same time an equal number of cases were treated merely with alcohol (two ounces a day), flavoured with a small quantity of quassia. The result was that the average duration of the cases, which were almost all quotidian, was:—

Under quinine treatment	...	...	...	$3\frac{19}{27}$ days
Under alcohol „	...	...	...	$3\frac{11}{27}$ „

Each remedy, you will observe, cured the disease, and the quina did so more quickly than the alcohol. The difference, however, was but a fraction of a day. Is alcohol, then, also a specific?

That most of the popular remedies for ague are of a tonic and stimulant nature—in fact, neurotic remedies—goes far to explain the anti-periodic action of quinine. As I said before, I do not dispute that quinine is the most valuable remedy we have for climatic fevers; but I do not admit any specific action, or that its beneficial effects in any prove the origin of these fevers in a specific poison.

To sum up briefly. Climatic fevers may be divided into two great groups; one caused by an excess, and the other by a deficiency of animal heat. But no specific poison is present

in either case. This, with regard to the first group, is scarcely disputed; while, as to the second, it has been shown that:—

1. Exposure in those localities, and at those times in which the cause of periodic fevers is active, necessarily involves exposure to chill.

2. All the effects ascribed to so-called malarious influence may be caused by the rapid abstraction of animal heat, without the intervention of any specific poison.

3. Exposure to chill is admittedly the cause of those diseases which are constantly associated with periodic fevers, as well as of recurrent attacks of the fevers themselves.

4. The effect of continued exposure to a high temperature is, at once, to depress the heat-generating powers, rendering the body more liable to chill, and to increase the susceptibility to the periodic form of climatic fevers. It also aggravates the intensity of the disease.

Under all the circumstances, it appears impossible to arrive at any other conclusion than that both the continued and the periodic forms of climatic fever arise from purely climatic causes.

CONTRIBUTION TO THE HISTORY OF THE EPIDEMIC FEVER PREVAILING IN THE COUNTRY DISTRICTS OF THE BOMBAY PRESIDENCY

DURING THE FAMINE-PERIOD OF 1876–78.

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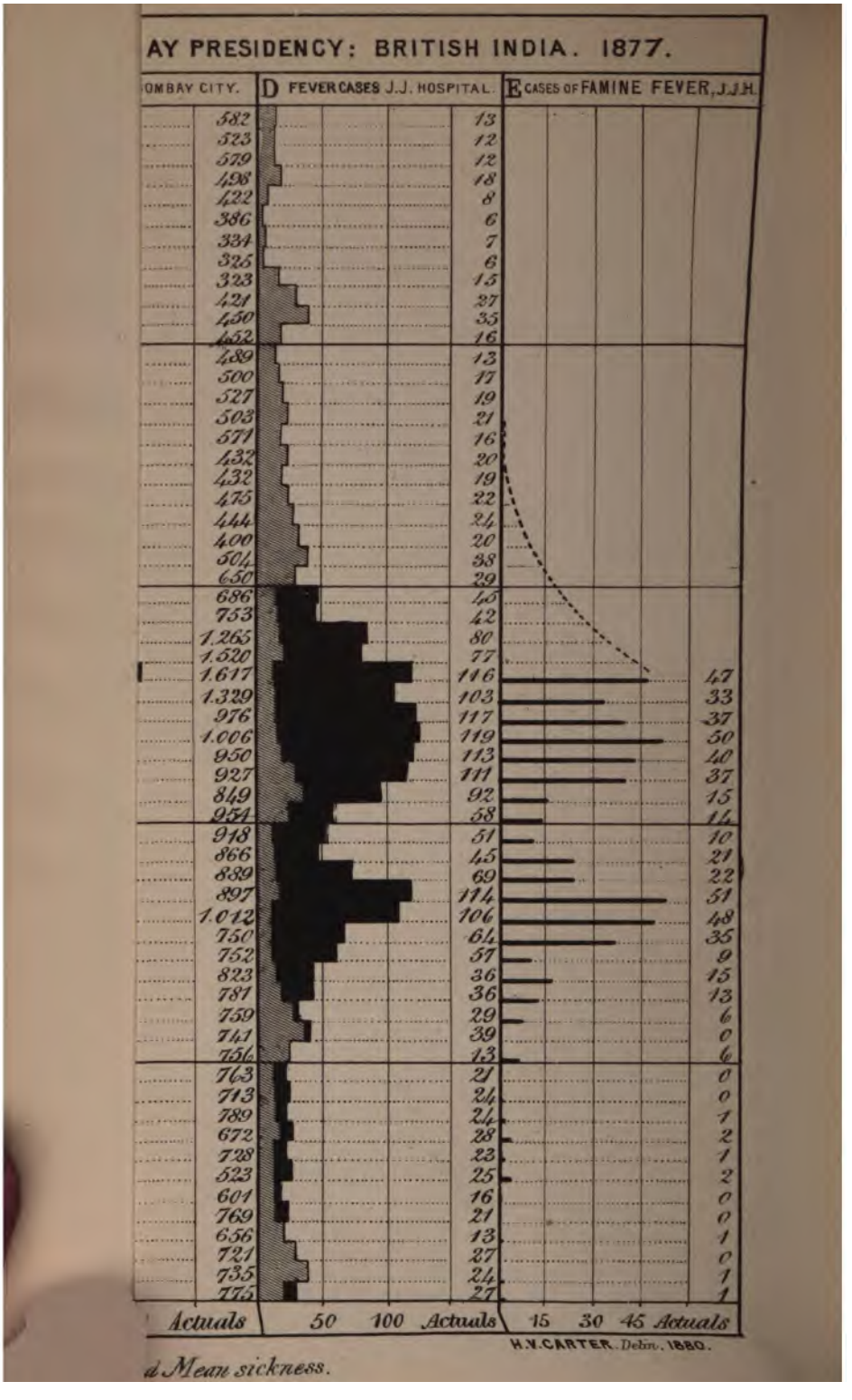
(*Read: February 21st*, 1881.)

THE sanitary and medical aspects of the late famine-sickness at Bombay are sufficiently important to engage the attention of epidemiologists in England, both of themselves and in the interests of our great Indian dependency.

Though I could not hope to add materially to the natural history of famine-fever, yet experience in W. India has not been without its special teachings; and this brief record (the only one of its kind) may at least serve to confirm European doctrine, and to stimulate and suggest renewed inquiry in India. Clinical details respecting the spirillum fever being here out of place, I desire it to be understood that all the conclusive evidence such details could afford is actually in existence; together with not a little collateral testimony, in favour of the epidemiological inferences herein submitted.

The numerical statements employed below are quoted from official statistics, published in the Government *Gazette*, and in the *Sanitary Commissioners' Reports* for the years concerned; other details being derived from the records of the native general hospitals under my charge from 1876 to 1880, or obtained through private sources, and from the public press.

The present Essay deals with but a part (and that the least investigated) of the late wide-spread famine-mortality in Western India, and may be considered as preliminary to the main work I hope soon to complete; yet there is a consistency in the data it embraces, which encourages me to offer it as it stands, in the conviction that epidemiologists acquainted with famine-fever, by either study or practice, will have no difficulty in arriving at a definite conclusion.


AY PRESIDENCY: BRITISH INDIA. 1877.
OMBAY CITY.
D FEVER CASES J.J. HOSPITAL.
E CASES OF FAMINE FEVER, J.J.H.
582 523 579 498 422 386 334 325 323 421 450 452
489 500 527 503 571 432 432 475 444 400 504 650
686 753 1,265 1,520 1,617 1,329 976 1,006 950 927 849 954
918 866 889 897 1,042 750 752 823 781 759 741 756
763 713 789 672 728 523 601 769 656 721 735 775
13 12 12 18 8 6 7 6 15 27 35 16
13 17 19 21 16 20 19 22 24 20 38 29
45 42 80 77 116 103 117 119 113 111 92 58
51 45 69 114 106 64 57 36 36 29 39 43
21 24 24 28 23 25 16 21 13 27 24 27
47 33 37 50 40 37 15 14
10 21 22 51 48 35 9 15 13 6 0 6
0 0 1 2 1 2 0 0 1 0 1 1
Actuals
50 100 Actuals
15 30 45 Actuals
H.V.CARTER. Delin. 1880.
d Mean sickness.

EXPLANATION OF THE CHART.

Columns A and B pertain to the present communication. The years 1875 and 1876 are normal years, and their numbers are entered in pale-tinted bars; the mean of these numbers being continued in the next three years for comparison, also as light-tinted bars. The black bars represent the surplus over normal mean, for these later years.

Column A.—The number of individuals on relief-works is the total present in last week of each month: the decline in February 1877, was incidentally only; the brief rise in September was due to renewed influx, when the rain seemed again to hold off. In 1878, the daily maximum of relieved amounted to 19,544; and in 1879, it was 17,656: further details are not available for these years.

Column B.—Total fever-deaths in nine country districts, the seat of famine per month, for normal and abnormal years, the numbers for 1880 not yet being accessible. The normal mean of fever-deaths is as follows, month by month, in order, from January, 8563, 7354, 8071, 8929, 9933, 9205; from July, 11,555, 12,441, 11,331, 10,023, 10,662, and 10,678; yearly total, 118,745: these are shown by the light bars in 1877, 1878, and 1879. Yearly actuals—1875, 119,910; 1876, 116,570; 1877, 223,388; 1878, 201,418; for 1879, the total is 129,840. Augmented mortality is seen to begin very shortly after the recognition of public distress, and it declined in general proportion with diminution of urgent want.

Column C.—Total fever deaths in Bombay city. The normal means for months in order were—January, 535, 511, 523, 500, 466, 409, 382, 400, 383, 405, 481; and December, 550; sum total, 5,545. Yearly actuals in order from 1875—5,244, 5,867, 12,832, 9 944, and 8,445. Augmented mortality is seen to begin very shortly after the recognition of public distress, and with the famine immigration then taking place: the maximum is in May 1877, when also relief was largest. Subsequently, fever implicated other classes than the Hindoo Deccan immigrants: fever is here "remittent fever" so-called.

Column D.—Admissions of "remittent" fever in the I. I. Hospital; no other heading being recognised for famine-fever. The normal means in order are—13, 14, 15, 19, 12, 13, 13, 14, 19, 28, 36, and 22; sum total, 218. Yearly actuals in order are—175, 267, 1,073, 639, and 273. Augmented admissions begin with recognition of public distress and immigration to Bombay, the maximum was a little later than that of Bombay town-deaths, because the hospital continued to be overcrowded, there being a limit to its capacity. Fever spread afterwards amongst many castes and races.

Column E.—Admissions of demonstrated spirillum fever, otherwise famine-fever, relapsing fever, or recurrent typhus: cases in the I. I. Hospital; there were as many others at the smaller hospital in April 1877, as here in May, and the earliest beginning of the disease is not settled. The correspondence with "remittent" fever admissions is striking, and particular months may be compared.

SUMMARY OF COLUMN B.

	Year.	Fever deaths.	Totals.	Excessive mortality in famine years.
Normal years	1875	119,910	236,480	188,326
	1876	116,570		
Famine years	1877	223,388	424,806	
	1878	201,418		
	1879	129,840		

Normal Data of Western India.—The presidency is mainly intertropical, extending over 125,156 square miles, and having a population of about 17,000,000. Excluding the province of Sind, its major part lies coastward, and rarely suffers from want of rain; but there is a large area of raised upland (known as the Deccan plain), where, as the country recedes from the western sea-line, the rain-fall gradually diminishes from 40 to 20 inches per annum, or less. It is here that the late famine occurred, the nine affected districts embracing a continuous area of 54,355 square miles, with a population of 8,000,000; of these, however, only five suffered severely, viz., Sattara (a part), Belgaum, Dharwar, and especially Sholapore and Kulladghi, which all lie between north latitude 14 deg. and 18 deg., and have an area of 33,873 square miles, with a population of 5,000,000, or about 147 persons to the square mile. Here the soil is mostly shallow, and derived from trappean rock; tree vegetation is sparse, and the incipient rivers comparatively small. Cultivation in this semi-torrid region depends upon rain-fall, which, in proportion as it decreases, is also apt to fluctuate in amount and distribution; the result being a nearly annual liability to variation in the harvest supply. Particularly is the meteorology of the last-named districts somewhat singular, owing to their position upon a slanting, shifting line where the two alternate monsoons of peninsular India may be said to meet, and become exhausted; and, either season failing, the land must suffer. Mean temperature of air 75 deg. to 80 deg. Fahr.; mean monthly range, 60 deg. to 90, or wider; mean humidity of year about 60 deg.; monthly means, 45 deg. to 86 deg.; minimum, often less; barometric pressure about 27.5 inches; the mean elevation being about 2,000 feet.

The inhabitants of the affected districts are mostly Hindoo Marattas, other races and Mussulmans being found to the east and south. A very small proportion of the Hindoos are strict vegetarians; but animal food is nowhere largely consumed. The great majority of the people are agriculturists, the remainder consisting of village officials, menials, and petty traders; there are very few large landholders, or manufactures, or large towns; and, as but one line of rail is available, transport is mainly effected by bullock-cart, and pack-animals. The population is habitually too poor to store either food or money; and marriage being religiously inculcated, families abound. The common dwellings are of clay, seldom of brick, not raised, and usually crowded; village or house-drainage is unknown, and the water-supply rarely selected. The people,

however, are not deficient in intelligence; they are industrious and abstemious, and preserve strict social discipline.

In average years the population is not unhealthy, and at least fully sustained: the normal death-rate may be not more than 25 per *mille*, per annum. The prevalent disease is "fever": "small-pox" and "cholera" are nearly endemic: "bowel-complaints" are common: under these four heads, together with "injuries" and "other causes", all the deaths are registered, according to a plan, now several years in operation, under native unprofessional agency. The returns are supervised by the Sanitary Commissioner and his assistants, who are commissioned Medical Officers.

Abnormal Events in the Deccan Famine Districts.—The localities named above were free from distress, and unusual disease, until the abrupt climatic derangements of 1876-77; and thence followed in succession drought, dearth, and disease. In the first of these years the rain-fall, beginning as usual in June, ceased generally in August, or long before the crops were grown; and in 1877, the same rainy season (south-west monsoon) was early interrupted, and not until the autumn was it supplemented by the north-east monsoon; so that for a second time the chief grain-harvests almost wholly failed, the loss being such as could not be at once made up, even with good years. After September 1877, heavy rain fell, but where not excessive, it was of little immediate avail; and the subsequent seasons of 1878 and 1879, proved not particularly favourable. The resulting calamities were especially felt in Sholapore and Kulladghi, where the total rain-fall in 1876 had been only 6.19 inches, and 4.76 inches respectively, or no more than one-fourth to one-sixth of the normal annual mean: hence, here, total failure of grain-crops, earliest and greatest distress, and the maximum fever-mortality. Similar contingencies, in like order, simultaneously ensued in the other districts named. In 1877, the general experience was of the same kind, and local distribution; and it was partially repeated during the spring and summer of 1878 and 1879.

Course of Distress.—1876. As early as August, apprehensions of scarcity were expressed, and prices of the staple grains began to rise; soon afterwards, relief works were established by Government in suitable localities, and, by the close of November, upwards of 150,000 labourers were collected together. Food was at famine rates, and, in some places, could hardly be procured at all; much of that offered for sale being old, and of bad quality. As pasturage and

agricultural operations, with other sources of remunerative labour, ceased to be available, emigration began on a large scale, and about the end of the year 13 per cent. of the affected populations had left their homes.

1877.—This period witnessed the severest sufferings, and descriptions have been published by eye-witnesses of the prevalent distress, in terms which it would be difficult to surpass. Cattle died of starvation, and the fields went untilled; there was no food but what was imported, and every sort of edible wild-plant was consumed; even water to drink was often scarce. The aspect of the parched land in the eastern areas was compared to that of a desert, with a sky of gloom.

Houses, or whole villages, were abandoned, and cherished personal ornaments were given up for coinage in enormous quantities, by the better class of ryots. Migration further extended, but many of the infirm, poorer, and low castes, would not leave their houses, though confronted with the prospect of death from privation. Although facilities for the importation of grain, and means of direct public aid were liberally afforded by the authorities, whilst private charity abounded, yet it seems to me impossible to concur in the view, that at this time only a few persons died of actual want; and how many thousands sank under the consequences of prolonged starvation has never been adequately estimated.

As the best available gauge of public distress, I have introduced into the appended chart the statistics of Government relief-works, at column A. In general there was a gradual increase of applicants for aid from November 1876 (beginning with 98,422) until June 1877, when the maximum was reached of 529,951, or about 10 per cent. of the entire population of the five worst districts; after this date, in prospect of the usual rains, the people were encouraged to return to their villages, and the works were closed in the ensuing November.

1878.—Distress augmenting in the spring, relief-works were again opened; the rainy season began late, and the fever-mortality continued to be excessive.

1879.—Distress was again prevalent before seed-time in the South Deccan and elsewhere (*e.g.*, in Kattiawar), "deficient harvest, owing to excessive and unseasonable rain, high prices, and the injury done to the later crops by locusts, grubs, and rats, all combined to bring about the recurrence of this calamity": quoted from the *Admin. Rep. Bo. Gov.*, 1878 and 1879. It is well known that in 1880 considerable suffering still persisted.

Sickness and Mortality.—1876. At the close of this year the deaths from fever were beginning to augment: see Chart, column B.

1877.—It is recorded that the total deaths in the Presidency this year were 627,708, or 259,448 (41.3 per cent.) in excess of those of the previous year, a remarkable augmentation, which was due mainly to "fever", the mortality, under that head, amounting to 336,865, or 116,032 in excess of 1876. Of this surplus, 106,032 (upwards of 90 per cent.) pertained exclusively to the nine famine districts under notice, there being elsewhere no such excess: within the affected area, the fever-deaths were 223,388, indicating an increase per *mille* of population double the normal, *i.e.*, 28.4, as contrasted with 14 per *mille;* and, in the two worst districts, the rise of fever-mortality was even higher, viz., from normal means of 13.1 and 11.4 per *mille*, to 40.4 and 50.5 per *mille* respectively. In the most easterly and poverty-stricken area of Kulladghi, where 9,757 persons died of fever in 1876, no fewer than 41,248 died from the same cause in the succeeding year; and this after a large emigration of families.

I should here remark that, amongst the 49,187 deaths occurring in the several relief-establishments, the casualties from fever, not being discriminated, could not be included above. I note, however, that a disproportionate part of these deaths (viz., more than one-fourth) occurred in the same district of Kulladghi alone.

On comparing columns A and B, it would appear that the fever-mortality did not rise so promptly as the public distress, lagging somewhat behind, as it were; this might be anticipated. Yet it should be remembered that there was a large emigration of people early in 1877, and a partial return of the same after June, when deaths soon became more numerous. A similar correspondence of this mortality, with movements of the population, is noticeable in the following year; and more emphatically was it established, as regards Bombay city, during the chief famine year; see also the fever admissions to the I. I. Hospital of both 1877 and 1878.

Striking as are these statistics, they do not show the entire loss of life referable to dearth fever; for, besides the hundreds of deaths of migrating villagers happening far from home, account should be taken of the many casualties everywhere due to the sequelæ of this disease, yet not included under the same heading. The official tables are necessarily silent regarding the amount of sickness not fatal; applying, however, my data from the Bombay hospitals, it might be said that for every fever-death above the mean, ten persons were ill.

Lastly, I note that whilst the high death-rate of the famine districts was due mainly to fever, yet "cholera" (asserted to be veritable), "small-pox", and "injuries", are returned as unusually fatal; moreover, there was an excess of deaths beyond the mean, amounting to 26,012 from "bowel complaints", and 68,460 from "other causes". Assuming that the three first-named headings had no essential connection with the dearth, it may be supposed that under "bowel complaints" would come famine-diarrhœa or dysentery, so widely fatal during 1877 in the adjoining areas of Madras. I find, indeed, that upwards of one-half the total deaths from bowel complaints in the whole Presidency, took place in the five worst famine districts alone; or a number higher than the total for the Presidency during any year since 1868. Particularly in Kuladghi the deaths from "bowel complaints" rose from 1,402 to 6,102, a proportionately larger augmentation than that of even the fever-mortality there; and in all the affected districts, the main increase from this cause occurred strictly within the famine period, or contemporaneously with the increase from fever. It has been conjectured that the term "other causes", embracing so considerable an excess, did this year include "privation", or starvation in its acute and chronic forms (under the latter coming exhaustion, anæmia, scurvy, dropsy); some such adjustment seems needed, but in the absence of information recorded when events were transpiring, discussion becomes futile.

1878.—Concurrent with persisting distress, was a continued high fever-mortality, the year's deaths amounting to 201,418, or upwards of 70 per cent. beyond the mean; it rose considerably after the rains, but also before rain in May. The distribution of surplus deaths this year underwent a remarkable change, for whilst the excess in the South Deccan declined, yet in the north districts, where the famine had not been very severely felt, there was now a considerable augmentation. I did not ascertain how this radiation of fever-mortality was produced.

1879.—It is well known that the fever-mortality continued to be considerable, but the Chart shows that after the first few months it declined to near the normal mean. Though public distress still persisted in parts, the wave of this particular sickness seemed to have subsided; and it is noteworthy that in some non-famine districts westward (whither was much migration), the fever-mortality of the year rose considerably.

Memorandum on the Cotemporary State of Provinces adjoining the Famine Districts of Bombay.—Within Presidency limits

the effects of the local dearth were extended (as would seem) only by migration of individual sufferers; and of such diffusion the instance of Bombay city is a remarkable illustration; there may have occurred others like it. In the nearer native states, however, towards both east and west, equal dearth with sickness prevailed, from the same climatic causes, during 1877: thus, in Mysore, the total reported mortality of 1876 being 54,265, during the first five months only of the following year it amounted to 85,915, an enormous augmentation, due chiefly to so-called "cholera", the fever-deaths (12,871) then also rising. Further data were not accessible, nor have I learnt any particulars respecting the state of the conterminous Nizam's territory.

With regard to certain districts of the Madras Presidency, not far separated from the worst affected in the Deccan, and, like them, suffering from defective rain-fall and want, it is recorded by the sanitary authority that whilst the mortality from famine diarrhœa was very great in the early part of 1877, yet at this time, when the fever-mortality was so high in Bombay districts and town, no cases were anywhere seen resembling famine-fever, and that the famine people in Madras did not show symptoms of fever of any kind. Whence, it appears that within an area, wide, but continuous, and uniformly afflicted, on the one side of a conventional mid-line, viz., the western, a fatal form of fever was very rife, whilst upon the other side, *i.e.*, the eastern, such disease was absent. Here, a positive datum being confronted by a negative one, the position amounts to discrepancy rather than contradiction; yet it is little satisfactory, however viewed, since it implies either error or omission; and assuming the general accuracy of official statistics, I cannot conceive any reconciling term, as regards the western area, except the supposition that famine-fever had been first introduced into the Deccan from Bombay city, and did not spread beyond Presidency limits. Such an idea is inconsistent with recorded dates and places of fever-mortality; yet I would not wholly reject it, were there valid evidence in its favour.

Appreciation of Events in the Mofussil.—Owing to absence of definite information in accessible records, I have to rely chiefly upon data collected in Bombay, and partly upon rational inference and analogy. The term "fever" in the Mofussil returns is held to mean (*a*) malarious remittents, and (*b*) pyrexia symptomatic of local inflammation; admittedly its application is very ill-defined, yet in the absence here of the "continued" fevers of Europe, and in the probable rareness of acute local disease, independent of malarious in-

fluence, the employment of this word for fatal febrile sickness seems hardly more inexact than the use of the expression "remittent" fever.

Normal Years: vide Chart, column B, 1875–76.—The rise of fever-mortality in and after the rains is comprehensible on the supposition that malaria then more abounds: and its persistence in the succeeding cold and hot seasons of the year may point to local diseases of the head, chest, and abdomen (*e.g.* cerebral congestion, bronchitis, pneumonia, hepatitis), variously amenable to climatic changes, age, sex, especially in the subjects of malarial cachexia. A previous long residence in the Deccan does not enable me to add more than that accurate data respecting "country fever" are needed at the present day more than ever; but the inquiry should be made a special one, and complete.

Famine Years: column B, 1877-78.—The old forms continuing in use, it was impossible that any new febrile disease, even if present, could have been specified, nor to meet the requirements of a grave sanitary crisis were fresh medical returns introduced: there remains, therefore, to search for indications in the available data, and by suitable arrangement of these the Chart becomes more than suggestive. Thus, it shows that prior to the famine was no unusual fever-sickness; and that with dearth the mortality rose so promptly as to indicate clearly a direct relationship; throughout, too, it was apparent that the surplus death-rate could not be a simple exaggeration of the previous normal state, but was more likely due to a superadded disease. This new sickness did not seem attributable to intensified malaria, because it began during an unusually dry season, at the end of the rainless autumn of 1876, and long before the scanty rainfall of June 1877:* and had there been present, any time during this

* At Kulladghi the rainfall in 1875 was 25.51 inches, and chiefly autumnal; the fever-mortality greatest from August to October (mean monthly deaths, 1,100, with narrow range), and least in February (562). In 1876, the rainfall was only 4.76 inches, and solely estival, yet the deaths from "fever" did not diminish as they should have, upon the hypothesis of contingent malarious influence; and, further, in spite of emigration and the continued absence of rain, they continued to increase throughout the first six months of 1877, rising from 948 to 3,776 in July, when only 5·3 inches of rain had fallen, or not more than the mean to date. During the corresponding period of the previous normal year, the rise was 705 to 953; and in no earlier record of these dates do I find any augmentation of fever-deaths comparable to that of 1877. In the first eight months of the chief famine year, with moderate rain, the deaths were 13,450 in excess of the same period of 1875 (normal year); and, as regard the dates from September to December, the large increase of 14,517 fever-deaths beyond the mean, seems hardly explicable by a distributed rainfall of 18.65 inches, even after making widest allowances for

period, even the ordinary degree of miasmatic influence, it is difficult to suppose the death-rate would have continued to rise with increasing aridity of soil. It is true that public distress was then augmenting, yet I am not aware that the destitute are specially susceptible to malarious influence, or likely to die therefrom in proportion to their destitution: yet were this so, the difficulty remains of accounting for so large a mortality, when actual miasm-production was at a minimum. That the increasing mortality in the hot season of 1877 was due to local inflammation, is an idea nowhere advanced; and, besides, local complications occur in fatal famine-fever nearly as often as in fatal "remittents", pneumonia, *e.g.*, being much the commonest in both infections.

After the rain-fall of September, severe malarious remittents possibly prevailed in the Bombay famine districts, as they are reported to have supervened in those of Madras, and persons enfeebled by want may have suffered unusually therefrom, with the result of an augmented mortality. At the same time the public distress did not subside, or at once diminish, with the advent of rain; on the contrary, it persisted, and the population of the districts becoming augmented by immigrants returning in hopes of a favourable season, which did not arrive, there were then more subjects liable to disease of all kinds.

1878.—Though the number of casualties was rather smaller, and their distribution in time, of necessity more uniform; yet the proportionate loss of life may have been hardly less than in the previous year. There is no better indication of malarial exacerbation, and the presence of spirillum fever was demonstrated in the Mofussil.

1879.—The fever-deaths now subsided, being excessive only during the early dry season of the year; there is no evidence of malarial diminution cotemporary with this subsidence,

the debilitating effects of want; and the more reasonable supposition would be that malaria had then aggravated pre-existing causes of disease, rather than originated a fresh source.

In an adjoining coast-district unaffected by drought, the deaths from fever rose with nearly equal rainfall to 10,662 in 1877, as compared with 6,452 in 1876, and the augmented mortality was attributed to the effects of malaria upon famine immigrants seeking relief in Kanara. Yet, if this were so, how is it that the mortality began with immigration before any rain had fallen? In March (a dry month) there were 836 fever deaths in 1877, as contrasted with 518 during the same month of the previous normal year.

In these notes I assume the ordinary theory of malaria to be borne in mind; combined heat and moisture being the *sine quâ non* of miasm-production. It is also presumed that the natural history of famine-fever is remembered: this disease being wholly independent of malarious influences.

and, as previously, public sickness in the famine districts and around (see a previous paragraph) seemed independent of seasonal influence.

Evidence from Analogy.—The malaria hypothesis not satisfactorily accounting for the late excessive sickness, experience of dearths in Europe or elsewhere on the globe might furnish the required explanation; and here the analogical inference becomes clear, being to the effect that cotemporarily with distress in the Deccan, there may have arisen a febrile epidemic disease, not malarious, but comparable to the famine-fever of other countries. An undefined pyrexia attendant on the last stages of privation has been mentioned in this connection; what it could be was worth testing. Fatal spirillum fever was highly variable at Bombay, and assuredly without great precautions and constant use of the microscope, the disease often would have eluded detection, even in hospital.

Direct Evidence.—It might seem impossible that a non-resident inquirer could learn the character of a comparatively fugacious complaint like relapsing fever, especially when he is separated from the nearer seats of the disease by a distance of 200 miles or more; yet the obstacles to proof are not insuperable, the following contingencies being possible.

(*a.*) A Deccan resident, while affected with "fever", might be brought in a few hours by rail to Bombay, and be at once admitted into hospital and be examined. (*b.*) The verbal testimony of patients showing specific fever might be accepted as to the date and place at which their sickness arose. (*c.*) Specimens of blood taken from "fever" subjects resident in the Deccan might be preserved and scrutinised at the Presidency town. As only by accident were new-comers brought to hospital, or came early of their own accord, instances like those under *a* especially would be extremely uncommon; and I consider it fortunate there are any of the kind to be quoted. Therefore, the following examples bear a significance which is not to be measured by their number. I regret that blood-specimens were not earlier procured from the Mofussil, but was not aware how to prepare them until the height of the epidemic had passed.

Proof under (*a.*) *Case.*—J. T. H., aged 35, Mussulmanee female domestic, was brought to hospital by the police on 30th of May 1877, from the adjoining Great India and Peninsular Railway Station of Byculla, where she was found to be in a state of delirium. "A friend with her states she has been suffering from fever for eleven days while at Poona (Deccan); she arrived at Bombay this morning to go to her brother's house, but became delirious, and, the friend not

knowing where to take her to, reported her condition to the police" (quotation from original notes of case). On admission, the axillary temperature was 102 deg.; pulse, 120 feeble; respirations 25, shallow; tongue dry, furred, brown; thirst excessive; cough and sibilant *râles* in chest. Next day, the blood was thus described by Dr. A. M. K.: "coagulation slow, plasmic bodies a few, white corpuscles many, spirilla seen." With prompt subsidence of the fever immediately following, jaundice and hepatic uneasiness were marked; she remained free from fever for nine days, when a pronounced relapse took place, lasting four days, on three of which the blood-spirillum was again found. Jaundice and delirium also returned; there was a prompt fall of temperature, preceded by the *perturbatio critica*, and much exhaustion subsequently. She remained in hospital eleven days longer, and then left in a convalescent state. I saw this woman frequently, and at the time noted there was no reason to doubt the truth of her history; how she acquired relapsing-fever at Poona is unknown; it may have been by contagion; and the case further shows that residents in the chief Mofussil town were in the way of catching the disease so early as May 1877: this is similar to experience in Bombay. See also another instance of fever conveyed by railroad in the following foot-note: in times, so much like a panic as those of 1877, native subjects are apt to fly abroad, and especially to the Presidency and larger towns, carrying with them, when sick, their malady so far as it is transportable.

Evidence under b. Case.—B. K., aged 20, country lad, Hindoo immigrant from the Deccan, destitute, but not starving, was admitted into the I. I. Hospital on 30th April 1877. History: had tramped from Poona in search of work and food; was seized with fever above the Ghâts (eighty or ninety miles by road from Bombay), and, resting two days, descended to Tanna (twenty miles off), again rested one day, and then walked to Bombay, when he was so ill that the day after his arrival he came to hospital, and was admitted into my clinical ward. At this date seven days had elapsed, according to his plain and repeated statement, since the fever began, five of which had been spent on the road: when seen on the evening of admission, he presented several usual symptoms of the new fever; temperature, 104 deg.; pulse, 108, small and soft; respirations, 28; bronzed facies, coated tongue, pains in the bones of the lower limbs, but spleen not visibly enlarged: the blood was found to contain many active spirilla. Next day, a marked crisis, the temperature falling 8 deg.; then a rebound to the normal temperature, and, ex-

cepting dysenteric diarrhœa with much debility, no interruption to convalescence: discharged 31st May. A common typical case of spirillum fever, seen late as usual, with no indication of relapse, except some periodic splenic fulness; the absence of a febrile recurrence not, however, impairing the patient's testimony in any degree; date of immigration about the acme of the Bombay epidemic, when crowds, like him, were arriving. I took occasion to point out to my class of students the great probability that this lad's fever had been contracted on the road, or possibly in his village home; his long journey on foot not, to my observation in Bombay, militating against this view: and the whole case, like that of the woman above, affords an illustration not only of spirillum fever prevalent in the interior, but also of its direct conveyance to the Presidency, where it might spread.

Case.—F. R., age 23, native Christian, carpenter, resident at Madras, set out thence on foot for Bombay; he was at Poona about a week ago (close of July 1877), and had fever; leaving, he had chills and renewed fever on the road; arrived in Bombay yesterday evening (31st), coming straight to hospital: then temperature, 104.4 deg.; pulse, 128. August 1st, pyrexia, dry skin, headache, pains, bilious vomiting, jaundice, constipation: blood examined upon admission, and also this morning, the spirillum found, though sparingly (Mr. S. A.), and again seen in the evening, when the temperature rose to 104.8 deg., next day declining to 97 deg. by critical fall; much weakness; discharged, after three days, at his own request. It could not be said whether or not this specific attack was the first, for the man asserted he had been suffering from "ague" for a month previously; if his statement were true, it in no way invalidates the significance of the case as one of specific fever acquired in the Mofussil, but also serves to show that particular means are needed to distinguish this peculiar form of pyrexia, which otherwise might not be recognised.

Case.—M. B., aged 35, Hindoo, ill-fed and anæmic, admitted with fever into the I. I. Hospital, 2nd July 1877. He gives the following history: left his native place in the Karav petta (famine district of Sattara) twenty days ago, in consequence of want of food; travelled on foot to Bombay, and arrived here four days ago, putting up at Omerkari; had fever on arrival in the town, and now comes to hospital: on admission was very ill; temperature, 105.6 deg.; pulse, 120; the pyrexia remitted in two days, and, on the third, subsided by crisis, the temperature sinking to 96.4 deg.; pulse, 95: there was some secondary fever with diarrhœa, and later on

two isolated paroxysms, which might represent the relapse. The blood was examined on the day of his entering hospital, and on the next also, the characteristic parasite being found on both occasions; it was not seen after the critical fall of fever.

Here, again, supposing reliance can be placed upon the simple statement of the patient that he had been in Bombay only four days, the clinical evidence would emphatically point to the conclusion that the specific fever exhibited by this man had been contracted before his arrival in the town. I have no hesitation in making this assertion, for whatever doubts be suggested as to the previous duration of his fever, the fact remains, that in accordance with large clinical and experimental experience, the incubation period of the disease would necessarily carry back the date of infection prior to the man's arrival. Were his attack a "relapse", the inference of extra-mural origin of the disease would be still more strengthened.

There are before me the notes of several other instances of demonstrated spirillum fever, dating back to day of arrival in Bombay, or to one or two days afterwards; and, in my own opinion, these, too, were valid evidence of the point in question, for no data are known tending to show that, amongst men, the contagion of this fever is capable of operating instantaneously in the production of pyrexia. On the contrary, in a remarkable series of six instances of reproduction by inoculation at autopsies of fever-patients which are intimately known to me, the periods of incubation were as follows:—three days, fourteen hours; three days, or possibly four days; seven days; seven days, four hours; seven days, four hours and a half: mean of the series, six days. As under ordinary conditions contagion is not likely to operate more quickly than by way of inoculation, it may be assumed these dates are not excessively long; and, therefore, any attack of fever supervening a day or two after immigrants' arrival in Bombay had almost certainly been contracted outside the town.

The epidemiological importance of this subject induces me to offer the following additional remarks, based on experience lately acquired in Western India.

I have shown by comparative experiment that relapsing fever is communicable at all times when the blood contains the characteristic bacterium; namely, during fever of invasion-attack (mean duration in man, seven to eight days), of first relapse (mean duration, four or five days), and even of second relapse (mean duration, two to three days), or for a total of about fifteen days in the course of a prolonged illness. But

these febrile periods are separated by apyretic intervals of about seven and nine days respectively; and, therefore, the entire time over which possibility of active manifestation of disease extends (and with this, the faculty of propagation) becomes enlarged to thirty days, or so; which is a term long enough for any journey a famine-immigrant would make in traversing the country from his native town or village to the Presidency, inclusive of due intervals of rest *en route*. No allusion is here made to the fact that the fever is communicable during an additional period corresponding to the few days immediately preceding visible pyrexia; and, lastly, I need only remark that a band of famine-immigrants once infected, might not only convey their fever for weeks together amongst themselves, thus allowing of even long sea-voyages becoming the means of transporting famine-fever, but by its propagation from their members to strangers coming into contact with the band, a still more prolonged manifestation of the disease becomes not only possible but quite probable. In fact, thus do epidemics arise, and thus the great significance of the few cases I have now submitted to notice; for it is evident that famine-fever once appearing in the Mofussil, wide-spread sickness was at least likely.*

* Amongst the many other confirmative data lately acquired, the following may be useful in this place. (*a*). It was matter of common observation to myself and other medical men that some immigrants entering the town were visibly ill, and had the aspect of low fever. (*b*). From a trustworthy official (non-medical) source intimately acquainted with the town of Bombay, I have the written statement that there were seen, in 1877, a considerable number of immigrants who were suffering from fever and prostration on their arrival in the town, the men and young children suffered most (a noteworthy remark, since here, as in Europe, mothers of families seemed to get famine-fever first, and then to communicate it to their associates, H. V. C.) Again, it is reported that at a station near Bombay where the immigrants were arrested in August 1877, "some of the children and a few of the men had fever"; this statement coming from a medical authority disallowing the existence of famine-fever at the time (date in question, 23rd of August). Again, in those quarters of the town where the fever was worst "cases like this occurred, a party, of say seven or eight immigrants, arrived with, say, three of them having fever; in a day or two there would probably be five or six of the same party attacked with the disease: there is no doubt at all of such cases having occurred". (*c*). What kind of fever the above might be is not obscurely indicated by the following instance belonging to my series of documents bearing on "contagion", but also applicable here (the Bala family). In August 1877, a woman and five of her children were admitted into hospital with fever, and it is stated that her husband was suffering from the same disease .... she furnished a history compatible with famine-fever, and had manifestly suffered of late from some severe illness; the five children all displayed the blood-spirillum, so that the nature of the case is beyond doubt. ... A native friend procured for me the following statement of these people. "They came from Satara (famine district); the man is a copper-smith; as they were starving, they left their home about two

Proof under (*c*).—Sholapore town and district suffered severely from famine and fever in 1877, and again in 1878. In May of this last year Dr. A. M. K. (who had taken part in

months ago (new moon of Jusht, native reckoning, H. V. C.), and removed to Poona (sixty miles nearer Bombay, H. V. C.), where the husband and eldest son (Mahboob) left the wife and young children, and came to Bombay. She remained at Poona for one month and five days, supporting her children by grinding corn on hire, and doing other occasional work. Fifteen days after her arrival in Poona, she became laid up with fever, when she remained ill for about a fortnight, when her eldest son Mahboob, hearing of her illness (? relapse, for, to my knowledge, native patients sometimes dated from first day of sickness, H. V. C.) came from Bombay and removed her with the children from Poona. She was at that time delirious, or in an unconscious state, and was brought by her son by the Great India Peninsula Railway train to Byculla (see the first case quoted above under (*a*) in the text, H. V. C.) They put up in Bombay in Peeroo Havildar's street, with a relation of theirs who is also a copper-smith (same caste of Mussulman, H. V. C.), and who supported them all for a time. A few days after her arrival here, her husband was attacked with fever: she does not know how many days ago, as she was at that time delirious. Shortly after, her eldest son was also laid up with fever. He was followed by his sister Auvarbee, who is about eight years of age, and is lying in the hospital, at present, in a state of collapse. After her, the younger sister Nyazbee, a girl of six, was also laid up with fever . . . . the next one attacked is the woman's eldest daughter, about twenty years of age . . . . About a week ago (when this account was taken down, H. V. C.) her son Dada, aged about ten years, was also similarly attacked . . . . last of all Illah-i, the infant at breast, got fever, and is very ill as yet . . . . The husband and eldest son are ill with fever at their home (the latter was admitted soon after and died in hospital with spirillar infection, H. V.C.) . . . . Their appetites have become abnormal, and they (especially the woman) continually crave for food: she and her young family have a peculiar musty and offensive smell about their persons; they are all much reduced in flesh, have vacant, staring looks, do not like to be questioned, and their temper has become a good deal irritable, and their mind somewhat deranged (expressions of native English, H. V. C.): the whole family is in a state of the utmost destitution." Such is the record, more of which need not be quoted here; and to some authorities it may seem the more striking, from its not having been collected for the purpose to which the present communication is devoted, but for different use, viz., to show how the spirillum fever might spread in a family. Having alluded to this subject of the contagiousness of the new fever, I may add that at the I. I. Hospital alone thirty patients sick with other diseases, fifteen ward attendants, and ten vernacular students were, during 1877 and 1878, attacked with demonstrated spirillar infection, and all within the medical wards alone where fever cases from without were only received. Experience at the G. T. Hospital was similar, if not more striking. No secret was made of these important facts; but, on the contrary, they were more or less known to the chief sanitary (local) and medical authorities in Bombay; and ample detail will be shortly forthcoming, together with other illustration of spread by contagion in this town. If in the town, then in the country districts; and hence, once more, the significance of the examples quoted in the text. It is a disadvantage that I cannot now treat of this topic more fully, but may add that every canon of contagion was complied with during the Bombay town-epidemic; and it would, indeed, be strange if experience in the Mofussil were different. Most unfortunately, the Presidency data were misinterpreted; but that circumstance does not render them null and void: it is only a serious sanitary warning.

inquiries at Bombay) visited, at my request, the above-named town and neighbourhood: he promptly reported in detail eleven cases of fever, unselected, finding the blood-spirillum in eight. In consequence of this information, I went to Sholapore after a few days, but was too late to confirm the observations; and not being able to remain away from the Presidency, had to return without fresh proof: however, upon examining the specimens of dried and stained blood which Dr. A. had prepared himself, I found the parasite still visible unequivocally in one, and probably in two other slides, which, like the majority, were too imperfectly prepared (from inevitable difficulties met with) to allow of thorough scrutiny. Of the cases, ten were Hindoos, five males, eight town-residents (one in respectable position), five being homeless beggars there; three were seen in villages near; several of the poorest gave a history of famine-suffering; all had symptoms more or less characteristic of relapsing fever, some describing the relapse; and this was so with the destitute woman J. S., whose blood furnished the decisive evidence: she was admitted a second time with fever (entered as being 'ague') into the Municipal Dispensary, and died there of dysentery ('bowel complaint'). Contagion in a family was indicated once: none of the patients had travelled to Bombay, and all denied having been in contact with travellers from a distance. I found the country parched up, and villages desolated, but saw few cases of fever: this is no matter of surprise, however, considering that the specific pyrexia is never continuous, and may be very brief. Evidence from the blood is equally contingent in practice, and a thorough investigation of relapsing fever may require a longer time, and more watching, than cases of ordinary malarious fever; not to insist upon special knowledge, skill and patience with the microscope. How readily the truth, regarding the nature of the late fever-sickness in the Mofussil might have been elicited on the spot, was shown by the result of the brief visit above described of a specially qualified agent.

Summary.—The late excessive fever-sickness undoubtedly followed upon dearth; the circumstances of its origin and early course indicate its non-malarious character; upon analogical grounds it might have been true famine-fever; and the presence in the Mofussil of relapsing or famine-fever (*i. e.*, of pyrexia with spirillar blood-contamination) was demonstrated in some cases. This combined testimony points to but one conclusion, which is not contradicted by other facts, and which legitimately explains all the chief phenomena of the epidemic.

ON SCROFULA, TUBERCULOSIS, AND PHTHISIS IN INDIA.

By JOSEPH EWART, M.D.

[*Read: April 6th*, 1881.]

Prior to the year 1840, we do not find much mention made regarding the existence of scrofula, tuberculosis, and phthisis pulmonalis among the indigenous population of India. This want of practical acquaintance with these pathological conditions was, probably, due to the imperfect utilisation of Laennec's system of auscultation and physical diagnosis; the extreme difficulty, in former times, of obtaining *post-mortem* examinations, in native cases; their frequent latency and liability to be masked by bowel complaints. In 1844 and 1845, Dr. W. A. Green proved the existence of phthisis among the natives of Howrah and Midnapore. In 1845, Dr. Edward Goodeve, whose recent demise was so touchingly commemorated in the President's address inaugurating the present Session of the Society, made similar observations among the people frequenting the civil hospital and dispensary at Cawnpore. It is remarked in the *Pathologia Indica*, published in 1848, by the late Dr. Allan Webb, that he had diagnosed phthisis among the Hindu race (Paharrees, hill-men) inhabiting the lower belt of the Himalaya range of mountains, and at Burdwan in Lower Bengal. In 1854, Dr. T. W. Wilson, in a thoughtful and elaborate paper, entitled "Tubercular Disease in the East", published in the *Indian Annals of Medical Science*, recalled attention to the subject, with a view to dispel the belief then entertained in England, regarding the supposed exemption of natives and Europeans in the East Indies from it. In 1856, Dr. Morehead (*Researches of Diseases in India*, vol. ii, pp. 394-419) relates his experience of phthisis pulmonalis, at the European General Hospital, Bombay, from 1838 to 1853; and among natives admitted into the Jamsetjee Jejeebhoy Hospital, Bombay, from 1848 to 1853. In the former, the admissions amounted to 184, with 79 deaths; in the latter to 445, with 268 deaths.

In my Address on Medicine, delivered at the Annual Meeting of the Bengal Branch of the British Medical Asso-

ciation, 1867-68, it was shewn that, during the ten years, from 1857 to 1867, 454 cases of phthisis occurred in natives treated in the Calcutta Medical College Hospital, of whom 285 died; that, from 1860 to 1867, 729 cases were diagnosed at the out-door dispensary of this institution, almost all of which took place among natives; and that, among Christians (a very mixed class of persons), during the decennium 1857 to 1867, there were treated, as in-door patients, 351 cases, with 139 deaths. A brief summary of sixty preparations, preserved in the College Museum, was also recorded in this Address, demonstrating the development "of tuberculosis in the lungs, larynx, trachea, mesenteric glands, subperitoneal connective tissue, intestinal glands (solitary and agminated), liver, spleen, arachnoid, kidneys and testes of natives of pure descent; and, also, of a few East Indians, who were born and reared in the country".

But there is good reason for the belief that the experience of developed phthisis, derived from hospital and private practice, affords only a partial criterion of the real prevalence of the tubercular diathesis, and disease in India. It not infrequently happens that grey miliary tubercles are discovered—sometimes in small, sometimes in large numbers, diffused, isolated, or in clusters—seemingly quiescent, and causing no great amount of local mischief, if adjacent emphysema be excepted; or the chalky remains of these and their consequent inflammatory exudations are found in the apices of the lungs, in the bronchial, or mesenteric glands of young natives who have succumbed to cholera, severe forms of specific, or malarious fevers, dysentery, or diarrhœa, and other acute forms of disease supervening upon malarious anæmia, and cachexia. In many of these cases, the history has pointed to repeated recurrences of malarial fever, with enlargement of the spleen, resulting in confirmed anæmia—in some, leucocythæmia, or leukæmia—succeeded, or accompanied by diarrhœa, terminating in asthenic, and unmanageable dysentery, but the lungs may never have been made the subject of complaint. In such cases, death may often be said to have been caused by intercurrent diarrhœa or dysentery before the tubercular diathesis had had time to end in active pulmonary consumption. It may, possibly, have been a consideration of such facts as these that induced the late Allan Webb, in 1848, to propound the doctrine that, at that time and previously, the reason why so many phthisical patients had been, and still remained, unrecognised, was that they were constantly carried off by diarrhœa, ending in dysentery; or by dysentery only, before the growth of tubercle had taken place in such

quantity in the lungs as to materially impair their functions, or to lead to their serious disintegration or disorganisation. He also, it may here be mentioned, regarded the hypothesis that, in this class of cases, the tuberculous material was diverted to the glandular structures of the intestinal mucous membrane, and, in great part, got rid of in the discharges, thus relieving proportionately the pulmonary organs. Be this, however, as it may, it is not difficult to understand how, in a person afflicted with the tubercular diathesis, inherited or acquired, bowel complaint may prove mortal without a single manifestation of its expression in the form of tubercle, grey or cheesy, being discovered in any part of the body after death, and how this constitutional proclivity sometimes, especially among the European soldiers, and their wives and children, and among the prisoners in the more overcrowded and unhealthy gaols, makes the dysentery of India uncontrollable or non-amenable to influence and action of moderately large doses of ipecacuanha.

Admitting all the above-mentioned facts elucidating the prevalence of phthisis among the various classes of the community in India, there is evidence, in the army returns, published by Bryden, calculated to show that phthisis is not nearly so prevalent among the native as among the European soldiers and their women and children. With the exception of Lower Bengal and Assam, in which the prisoners are still, it is feared, greatly overcrowded, this observation is upheld in drawing a corresponding comparison between the gaol population and the British soldiers, their wives and children. The figures I am about to quote, in support of these general statements, embrace six years, 1871-76.

Thus, the ratio of admissions per 1,000 of strength with phthisis amounted to 9.0 among the soldiers of the European army of India, to 8.8 among those of Bengal, to 10.8 among those of Madras, and to 9.0 among those of Bombay; to 9.6 among the women of the European army of India, to 10.4 among those of Bengal, to 8.0 among those of Madras, and 8.7 among those of Bombay; to 8.3 among the children of the European army of India, to 8.2 among those of Bengal, to 8.7 among those of Madras, and 8.2 among those of Bombay.

According to Professor Aitken,* the ratio varied in the American army, during 1861 to 1863, from 8 to 9 per 1,000, and in the British army, during 1861 to 1862, from 9 to 10 per 1,000 of mean strength.

In the native army of the Bengal Presidency, the corresponding ratio, during 1871 to 1876, amounted to 2.5, in that

* *Practice of Medicine*, vol. ii, p. 846, sixth edition.

of Bengal proper and Assam, to 3.9, in that of the Gangetic provinces, to 2.9, in that of Rohilcund and Meerut, to 3.0, in that of Agra and Central India, to 2.6, in that of the Punjab, to 1.6, in that of the Punjab frontier force, to 2.1, and in that of the Central India irregular force to 0.8.

For the same period, in the gaol population of the Bengal Presidency, the ratio of admissions per *mille* with phthisis amounted to 4.2, in that of Lower Bengal and Assam, to 8.9, in that of the Gangetic provinces and Oude, to 3.2, in that of the Central provinces (excluding Saugor and Jubbulpore), to 1.5, in that of Agra and Central India to 2.1, in that of Rohilcund and Meerut to 2.8, and that of the Punjab to 2.1.

These results warrant the inference that phthisis is much less prevalent among the native population of India than among the inhabitants of these islands, or among the British soldiers and their families located in India. And a comparison of the rate of prevalence among these latter classes, with that of their *confrères* quartered in the United Kingdom, shows that a transfer to India in no way materially diminishes the admissions from this mortal disease.

The figures used here only give arithmetical expression to facts which have long been known to the clinical physician in all parts of British India. One of the first things which strikes him, when contrasting his home with his Oriental experience, is the preponderance of abdominal disease among his patients over affections of the thoracic viscera. The comparative infrequency of tubercular disease of the lungs forms no exception to the predominating rule, even when full allowance has been made for latent and undiscovered tubercle. It is quite rare and exceptional to hear of whole families, or even of several members of a native family, being carried off by phthisis. White swellings of joints, and strumous enlargements of the glands, or cicatrices, resulting from suppurating scrofulous glands, are comparatively infrequent. To contribute some partial explanation of the facts hereinbefore advanced, as far as our knowledge will admit, and as briefly as may be consistent with doing justice to the subject, will be the object of the subsequent observations.

1. *The Influence of Fresh Air and Living much in the Open Air.*—In the *Lancet* of the 6th of January 1866, p. 20, Dr. Henry Bennet wrote as follows:—"Singularly enough, I believe, I am the first, and as yet the only writer on climate, who has recognised and forcibly insisted on the all-important and self-evident fact that consumptive patients should reside, winter or summer, in England or abroad, where they can

breathe pure air, night and day, that is, in the country in healthy villages, and in the healthy outskirts of towns. Their breathing pure air is of infinitely more importance than a few degrees of temperature more or less, or a little more or less protection from this or that wind. A fact so consonant with modern physiology and pathology has only to be brought forward to be universally acknowledged, and the time is near when medical men will wonder how they could ever think of cooping up patients in unhealthy southern towns for the sake of mere warmth." Doubtless, much of the exemption from consumption enjoyed by the natives of India is largely due to the general conformity with the principles herein inculcated. By far the greatest proportion of the people are engaged directly and indirectly in the cultivation of the soil. Out-door occupation is the prevailing rule. The huts and houses in which they dwell, whether in villages, towns, or hamlets, are full of crevices, chinks and openings, so that anything like the overcrowding and breathing of air which has already been utilised in respiration is not nearly so marked as in colder latitudes. The mildness and warmth of the climate during the greater part of the year renders a breezy circulation of air,—against which the habitations, the sleeping rooms especially, in frigid and temperate climates are religiously constructed,—in tropical regions welcome, healthy and antiseptic. Cellar life, or the living in rooms below the level of the ground, even in the largest cities, is unknown. The ingress of air from without, and the egress of it from within, native dwellings, in town and country, is uninterruptedly going on, night and day, by natural ventilation, with a windy and draughty freedom made agreeable to the people by a generally warm atmosphere. This is a very different state of matters when compared with the stuffy, close, and ill-ventilated bedrooms of this and other temperate countries. Both in towns and in the country, a considerable portion of the twenty-four hours is passed practically in the open air, in open houses, rooms, courtyards, verandahs, or in the fields. The respiring of air, which has already been recently breathed, is thus in great part prevented. And, though the air must often be loaded with impurities derived from the insanitary condition of most of the cities, towns, and villages of the country, yet, owing to free perflation and ventilation of native dwellings, its contamination with the organic products of respiration is, in large measure, reduced to a comparative minimum. This source of the ill-health and impoverishment of blood, conducing to scrofulous and tuberculous dyscrasia, is considerably minimised in India, and

something of the diminished liability of the natives to consumption must be ascribed to the greater freedom of the air breathed from the noxious products of that which has already been respired.

2. *The Influence of the Food Chiefly Consumed.*—Although, as was pointed out in my work on the *Sanitary Conditions of Indian Gaols*, there are no exclusive vegetable feeders among the peoples of India, it must, nevertheless, be admitted that, in all classes, and more especially in the case of the Hindus, the dietaries are mainly constituted of vegetable aliment. Among the population inhabiting the low-lying and alluvial lands of Bengal, Assam, and near the sea coast in Madras and Bombay, and in the vicinity of rivers and lakes in the interior of the country, the staple article of consumption is rice, affording, with a small proportion of nitrogenous, a large excess of the carboniferous aliment. The latter is supplemented by the Hindus by means of milk, dhal, or curds, fish, fowls, etc., but principally by the various species of dhal and other leguminous seeds, rich in nitrogenous matter. Fruit, ripe or unripe, and dried and green vegetables are universally consumed. The cooking is excellent, and the seasoning of the food with spices and condiments irreproachable. Abundance of good salt is now available to the poorest as to the richest classes of the community. In the uplands of the aforesaid provinces, in the North-west, Punjab, Central Provinces, Deccan, Rajputana, the staple article of diet is wheat, whilst in Madras it is raggy (elucine corocana), cholum (holcus sorgum), chumboo (holcus spicatus), all of which are rich in nitrogenous nutriment. In these parts rice is a luxury, and mainly used by the well-to-do. Dhal, also, is not so much consumed as it is in those provinces where rice is the chief element of the food. Milk and curds, fish and fowls are abundantly partaken of. Oleaginous aliment is represented by melted butter, or ghee, the oil of the cocoanut, olive, linseed, mustard, etc. The Mahomedans consume more flesh meat than the Hindus, such as kid, fowl, mutton, and beef. The bread made from wheat and other highly nitrogenised grains includes kernel, and a large quantity of husk, and is unleavened, and baked in the form of chupattie (a small and thin cake). On the whole, it may be roundly said that, among all classes, wholesome vegetable aliment, presented to the stomach in an easily digestible dress, constitutes the leading feature of the indigenous dietaries, whilst, in a much smaller proportion than is the case in this country, food of animal origin is eaten. Here the relative proportion of the nitrogenous and non-nitrogenous nutriment is

somewhat reversed. There is too much of the former, too little of the latter, and it is probable that the quantity of oily food of the most digestible kind is deficient in the dietary of those classes most liable to scrofula, tuberculosis, and phthisis. With such simply constructed dietaries as those used by the people of India, the proteine and albuminous principles are represented in quantity and quality suited to them and the climate in which they form the permanent population, whilst the amylaceous, saccharine, and oleaginous principles exist—it may be—in such an undue proportion as to favour premature degenerations of a fatty rather than of an albuminous nature. If there be a germ of truth in this part of my explanation; if the mild and comparatively unstimulating nature of the food employed by the people has something to say in lessening the proclivity to tuberculosis in India, then it may be assumed that, coupled with the general temperance in the matter of alcoholic liquors, it has, in the course of the several thousand years, during which it has remained practically unchanged, as to its nature, quality, and mode of preparation, exercised a potent influence in preventing the acquirement, and in restricting the inheritance of the scrofulous diathesis. Nor should the probable protective power of the prevailing custom of the protracted suckling of the young, often beyond the completion of the first dentition, be omitted from consideration.

3. *The Influence of High Temperature in Lessening the Frequency and Mitigating the Severity of Bronchial, Pulmonary, and Pleuritic Disease.*—Although coryza, bronchitis, pneumonia, and pleurisy may be seen in the autumn and cold months as primary affections, they are, particularly the three last-mentioned diseases, most frequently met with, in an asthenic form, as sequelæ, or complications of malarious and other fevers. The cold bath daily had recourse to by almost all classes, high and low, affords a great protection against colds, nasal, and bronchial catarrhs. Moreover, the skin of the natives being so much directly exposed and regularly subjected to inunction becomes inured to withstand, to an extraordinary degree, vicissitudes of temperature; still, in the autumnal and cold seasons, when the thermometric range is at the greatest, and when the radiation of heat from the surface is extreme, the advent of these affections is favoured. But, it may be formulated that, owing to the usually high temperature of the atmosphere, the inflammatory diseases of the mucous membranes of the air-passages are less potential in exciting tuberculosis of the bronchial glands and pulmonary parenchyma than in frigid and temperate latitudes.

Even in the cold months, when these complaints mostly prevail, they seldom succeed in lighting up, as they so commonly do here, tubercular disease in the apices of the lungs. The reason of this comparative exemption is probably to be found in the absence of the tubercular diathesis—acquired, or inherited. In disposing of this part of my subject, not only the mildness of an Indian winter, which, in most parts of the plains, is much warmer than our warmest summer in England, but its shortness, varying from two to four months or so, must be borne in mind.

4. *Influence of the Diminished Prevalence of Measles, Whooping-Cough, Scarlatina, Mumps, etc.*—The influence of these diseases in the production of scrofula is widely acknowledged. The two first and last exist in India; but to a much smaller extent than in Europe. Moreover, when measles do occur, the catarrhal and bronchial mischief is much mitigated by the warmth of the climate. Scarlatina has, I believe, only been recognised in a few isolated instances, as imported into Bombay or Calcutta by canal steamers, but it has not been known to spread as an epidemic. The red fever, described by Goodeve, and the dengue, pictured by Charles, are not accompanied by any special nasal or bronchial catarrh. It may fairly be claimed that we have in the considerable exemption of the native children of India from these scrofula-generating diseases one very important reason why tuberculosis is less frequent in India than in Europe.

5. *Influence of the Infrequency of Scrofula.*—Partly on account of the general great freedom of the native children from those diseases of childhood which are potential in developing scrofula, this, the parent of much tuberculosis, is not so commonly met with in India as in this country. That this is the correct explanation is countenanced by the fact that the diarrhœa of native children is not very frequently followed or accompanied by tabes mesenterica, or cheesy infiltration of the mesenteric glands. Webb, as I have previously remarked, believed that such diarrhœas were a medium for the elimination of tubercular matter from the system. But do we find that, though tainted matters may be expectorated during bronchial catarrh, in a child or youth of scrofulous diathesis, or actually suffering from confirmed phthisis, the growth of tubercle is, as a general rule, materially diminished? Do we not rather discover, in most instances, that with each succeeding bronchitis, the tuberculosis has extended? It may, however, be argued that the cases are not quite parallel, inasmuch as the products of scrofulous inflammation of the bronchial mucous membrane, or of the pul-

monary parenchyma, cannot be so easily ejected; and that tainted endothelial elements are more likely to be absorbed by the interlobular lymphatics and capillaries, and thus tend to aggravation rather than relief, than in the case of the intestinal mucous membrane and its adenoid bodies, where, owing to exalted peristalsis, all abnormal products are more rapidly expelled. If, it may be contended, as sometimes happens, a localised cheesy mass may be successfully broken down and extruded from the apex of the lung, and followed by a certain measure of repair and cicatrisation, why should not this process much more frequently succeed when the scrofulous inflammation is concentrated in the glands of the mucous membrane of the intestine, provided with far greater facilities, than are the lungs and bronchi, for the speedy escape of the offending matter? But if, as may be conceded, such a termination may possibly be more frequent, in the case of tubercular solitary or agminated glands, there is no good reason for believing that it takes place so commonly as to account for the comparative immunity of the youths of India from scrofula, tuberculosis, and phthisis. In these glands, as in the lungs, the process of disintegration is slow and tedious, repair difficult and exceptional, and, during the inflammatory and ulcerative process, the rule is to implicate the lymphatics and mesenteric glands, and to cause their infiltration with scrofulous material. But, as has been observed, this is seldom the result of the diarrhœa of native children and youths—much less frequently than one would expect, were it associated, in its initiation, causation or course, with the scrofulous or tubercular diathesis.

The old theory of a supposed antagonism between tuberculosis, or phthisis and malaria, has been abandoned. That phthisis occurs in malarious localities has been sufficiently well proved. If, in India, the diathesis favouring its development is not so prevalent as in many non-malarious countries, this comparative immunity need not be assigned to any active antagonism between it and malaria, but rather to the almost universal respiration of air, in an unusual degree, uncontaminated with the organic and other products of that which has already been breathed; to the antiscrofulous nature of the food consumed; the existence of an elevated air temperature, which is, in a great measure, antagonistic to inflammatory diseases of the air-channels and cells, and conducive to easy recovery from them when they do supervene either in their primary or secondary forms; very great freedom from those exanthemata and other diseases of childhood which, in other countries, are so often accompanied or succeeded by

strumous affections; and to a somewhat exceptional immunity from the scrofulous diathesis. Still, seeing the immense amount of extreme anæmia and cachexia caused by malaria, and a remarkably disproportionate prevalence of tuberculosis, side by side, the natural inference is that, though the poisons may not be antagonistic, there does not seem to be much in common between them. But this, to my mind, is only another of the several proofs given in this communication that either the scrofulous diathesis is altogether absent, or so completely dormant in a vast majority of native children and youths that even the tropical anæmia and terrible cachexia of prolonged malarial disease are incapable of leading to the development of tuberculosis or phthisis in either corresponding degree or frequency. When, however, on the other hand, this proclivity exists as an inheritance, or has been acquired, as in European soldiers, and their families, in Eurasians or half-castes, and in native convicts in the overcrowded prisons of Bengal, Assam, and the Gangetic provinces, malarial cachexia does not oppose, but may be rather said to facilitate the advance of tuberculosis and phthisis.

Before drawing these observations to a close, I propose to answer, as far as my experience will justify my doing so, the question whether longevity is promoted by transferring persons predisposed to, or suffering from, tuberculosis or confirmed phthisis from this and other temperate latitudes to the Plains of India. There is an impression that persons born of phthisical parents, but in whom there is no tangible evidence of tubercular deposit in any part of the body, do better in that country than at home. They certainly escape, to a marked extent, the frequent attacks of bronchial catarrh, pneumonia, and pleurisy, which are rightly associated with the excitement of tubercular activity in the bronchial glands and the pulmonary parenchyma. The greater exemption of children and youths from measles, scarlatina, whooping-cough and mumps, is also an additional advantage. The warmth of the climate, the free action of the skin, so long as it is not excessive or profuse, and the increased expansion of the chest-walls to compensate for the rarefaction of the air, are all favourable. But these advantages are counterbalanced by the great heat and malariousness of the plains, which, even in those individuals who may have been fortunate enough to escape recurrent attacks of paroxysmal fevers, eventually lead to anæmia and debility, conditions which are favourable to the development of tubercle in those diathetically predisposed. These advantages are still more powerfully counteracted by repeated invasions of intermittent fever, and season-

ing febrile affections, ending in malarious cachexia and dyscrasia, and defection in the power of generating animal heat. Add to these a predominating proclivity to bowel complaints, and the difficulty of obtaining, in such an exhausting climate, sufficient exercise to perfect function, and to confer tone upon the nervous and muscular systems, and then not much will remain to be said in favour of advising those descended from consumptive ancestors to exchange a career at home for one in any part of the plains of British India. Cases may be cited which have done well, particularly in the drier climates of Mysore, some parts of Rajputana, of the Central Provinces, of the North-West Provinces, and the Punjab, but such exceptions are not peculiar to India, and I do not find that these occur more frequently there than in England, or in other countries where phthisis prevails, and is a most constant and important contributor to the bills of mortality.

When there is unmistakeable evidence of tubercular growth in the apex of either or both lungs, or when phthisis has become pronounced, clinical observation of Europeans so affected clearly leans to the view that they derive no benefit whatever from residence in India. The causes already mentioned, which are potential in exciting tuberculosis in those persons inheriting a scrofulous diathesis, operate with redoubled energy and effect where tubercle, whether hereditary or acquired, has been formed. Rightly interpreted, they go far to explain why, in such cases, the tendency is rather to progressive augmentation of the tubercle in the lungs than to its arrest or diminution, with or without, as the disease advances, tubercular ulceration of the solitary glands of the large and small intestine, and of the agminated glands of the ileum.

REPORT BY LÉON COLIN ON THE DEATHS OF ESQUIMAUX FROM SMALL-POX, IN PARIS, IN JANUARY 1881.

By WILLIAM SQUIRE, M.D.

[*Read: April 6th*, 1881.]

On September 26, 1880, a troop of eight Esquimaux arrived at Hamburg from Labrador to exhibit themselves in Europe. They visited Berlin on October 18th, staying a month there. On November 29th they left for Prague, and stayed there from 20th to 30th of November. They were at Frankfort from the 1st to 11th of December; at Darmstadt from the 13th to 18th; and at Crefeld from the 18th to 30th of December. Small-pox was prevalent at Prague. A fortnight after leaving, on December 14th, one of their number, a girl, died with the eruption of small-pox on her. A fortnight later, December 27, at Crefeld, a woman died of hæmorrhagic small-pox before the eruption appeared. A child was seized at this time, and died on the 31st. She was left at the hospital on December 30th, when the other five members of the troop started for Paris. The Burgomaster of Crefeld at once telegraphed to the Prefect of the Seine: "Five Esquimaux, with attendants, will arrive in Paris on December 31st at a quarter to five A.M., from Liege; they left a sick child here, now found to have small-pox." On the 1st of January, the five Esquimaux were vaccinated with animal vaccine from tubes, but no effect was produced, nor was a second vaccination tried until five days later, when two of them were already sick. All five were down with small-pox by the 8th of January, and were sent to St. Louis on the 9th, two dying before the eruption, with high temperature, and hæmaturia; the others had the complete rigors of hæmorrhagic small-pox, and, before the 16th of January, all had died. The first to show signs of sickening were a little girl and her father; next, on the 6th of January, the husband of the woman who died on the 27th of December at Crefeld; he also dying with hæmorrhagic small-pox, and no eruption. The mother of the little girl, who sickened first in Paris, was seized on the 8th of January,

and died on or before the 16th. They were all infected, either from the woman who died at Crefeld on the 27th of December, or from the child left behind ill on the 30th of December, probably from the woman, as the child's illness was a day or two later than hers. Both these were infected by the young girl who died at Darmstadt on the 14th of December or by contagion emanating from her.

THE "WOOLSORTERS' DISEASE", OR ANTHRAX FEVER.

By JOHN SPEAR, Esq., Medical Inspector to the Local Government Board.

(*Read: May 4th*, 1881.)

Mr. President and Gentlemen,—When I was asked by the Council of this Society to read a Paper here on the subject of the "Woolsorters' Disease", I hesitated to accept the honor; for my Official Report to the Local Government Board on the results of my investigations was soon to be published, and I felt I had no right to occupy the time of the Society with matter, the substance of which, perhaps before I could communicate it to you, would be found elsewhere. But my friend Dr. Thorne told me that the Society wished to have the subject of anthrax—for that is the disease—a subject almost unknown in the annals of British medical literature, formally before them, so that if need be it might be discussed; and this being so, I of course gladly and gratefully accepted the task.

Anthrax, notwithstanding the obscurity I have remarked upon as still surrounding it in the human subject, is a disease of greatest antiquity. It is believed by certain historical writers that the malady which constituted one of the plagues from which the Egyptians and their cattle suffered at the time of Pharoah was of the nature of anthrax, and it is certain that the ancient Greek physicians describe its external form, under the name it still bears. Between the fourteenth and eighteenth centuries, according to Bollinger, its various forms in the lower animals, and so far as they were known in man, were confused by writers under different names; until Chabert in 1780, brought forward numerous and cogent arguments to prove their unity. During the present century the disease in animals has been much studied, especially in France. At first the question of its contagiousness was stoutly fought over, for there were not wanting high authorities, amonst them Virchow and Heusinger, to advocate its malarial origin; whilst still others, following Delafond,

regarded it as a disease of over-feeding in sheep and cattle; and this latter view was a favourite one even within the last few years amongst our own veterinarians.

In 1855 Pollender announced his discovery of the constant presence in the blood of suffering animals of "countless rod-like bodies". Brauell quickly followed with a like observation, and Davaine a few years later pronounced these bodies to be bacteria, of vegetable nature. He said, referring to the disease in sheep and cattle, that these bacteria or their germs might be found in every case of anthrax; that their appearance preceded the development of the morbid symptoms; that their number in a single drop of blood was sometimes from eight to ten millions; that he had reproduced the disease in other animals by the inoculation of a millionth dilution of such a drop; that the infectiveness of the blood disappeared with the disappearance of this bacterium in the progress of decomposition; and, finally, he claimed to have shown that this organism constituted the poison of the disease. There have been many most exact and faithful workers in this field since that time, and this conclusion has not been shaken; on the contrary, it is placed now, it may fairly be claimed, beyond the range of hostile criticism—the bacillus anthracis is the contagium of anthrax.

Although these observations demonstrated the transmissibility of the disease from one animal to another, they did not altogether disprove the theory of its spontaneous origin, under ordinary circumstances of its development, in the soil; and it was still looked upon by many as essentially a disease of malarial nature. There were many observations that seemed to support this view. There was the geographical distribution of the disease which corresponded with that of malarial fever. It was proved almost as clearly as it is proved for malarial fever that a damp soil rich in vegetable matter was the constant characteristic of anthrax districts; and that those unfavourable conditions of soil removed, anthrax as an epizootic or enzootic, like intermittent fever, gradually disappeared. It was shown also that the seasons in which the disease most prevailed amongst individuals corresponded with the seasons of greatest prevalence of malarial fevers amongst men. On the other hand there were many swampy districts that were free both from malarial fever and anthrax, and it was found there were several striking exceptions to the rule that the two diseases prevailed in the same locality—observations suggesting that something more than a marshy condition of soil was necessary for the development of both diseases, and that this determining factor was not in both

cases one and the same. Then, several outbreaks were recorded that were clearly dependent upon contagion; and, again, it was found that in certain districts, and on certain farms (no change in the condition of the soil being apparent), when precautions were taken against the spread of the disease, and especially when the bodies of animals that had succumbed to it were carefully removed, instead of being left upon the ground or buried in shallow graves, that its former prevalence ceased. In 1869, a Russian Commission that had been appointed by the Government to inquire into the enzootic prevalence of anthrax in certain provinces of the empire, reported this careful disposal of carcases as one of the most necessary of precautionary measures; and it has gradually come to be accepted by authorities, as it will some day perhaps be generally recognized of malarial fevers, that the state of soil and atmosphere remarked upon is only operative in so far as it offers favourable conditions for the retention, and perhaps for the growth and development, of a specific contagium. It is not improbable indeed that such conditions—a nidus rich in vegetable matter, together with warmth and moisture—are, to one or both these contagia, indispensable to certain stages of their development. This view, it may be added, as to the auxiliary influence of the soil in the prevalence of anthrax, is strengthened by recent experiments of Pasteur and others, by which it appears that the earth around the buried carcases of infected animals may contain bacilli, and be capable of transmitting the disease.

Whilst these somewhat complete observations were being made upon the disease in animals, cases were from time to time recorded, from anthrax districts, of infection in man. But these cases were mostly of the external form of the disease—the malignant pustule; records of attacks of the primary constitutional infection of what I have designated anthrax fever—were few and far between, are incomplete, and often not unequivocal. How little indeed was known of this form of the disease may be gathered from the fact that Bollinger in his Article on anthrax in Ziemssen's Cyclopædia, the best treatise on the subject in the English language, speaks of it as an almost necessarily fatal malady, and states that one case only of recovery—a case of Leube's—is on record. In short, some of the most important phenomena, as it seems to me, associated with these attacks, and some of the most interesting forms of the disease, have, so far as I can find, not yet been recorded. Perhaps, occurring as they must do, most usually in malarial regions, many cases have been looked upon as forms of malarial infection. For when I come to

describe the disease as I have studied it in the woolsorters, many, no doubt, of the distinguished members of this Society who have made malarial fevers their study in tropical regions, where pernicious and irregular types are more commonly met with, will recognize close clinical and pathological affinities between the two affections, as well as the etiological relationship that appears to have misled the older observers.

But before proceeding with this part of my subject it will be convenient to give as briefly as possible a *resumé* of the history of the "Woolsorters' disease", and of the results of the investigations which demonstrated the identity of this disease with anthrax.

The woolsorters of the Bradford district are numerically a small body of between two and three thousand men. Their occupation, which is looked upon as a skilled one, and requires a long apprenticeship, consists in sorting or dividing the wool or hair, that constitutes the raw material in the worsted manufacture, into several different qualities. The material thus manipulated is largely of foreign growth—the fleeces of Turkish goats, of Persian sheep, of the South American alpaca, and camel hair from Russia and China, and this, unlike English wool, is mostly unwashed, and is dry and dusty when it reaches the sorters' hands. Afterwards, it comes to be well scoured, and machinery then largely takes the place of hand labour. Now for more than forty years, ever since this foreign wool was to any extent used, it had been an article of faith amongst the sorters that those who had the handling of it in this early process of its manufacture were liable in consequence to a mysterious and often fatal malady; and believing this, and finding that the fact was not recognized by others, the matter became one of absorbing interest to them, and they watched the behaviour of the disease—the circumstances of its attacks and its symptoms—with minute attention and often extraordinary intelligence. The knowledge the sorters themselves thus acquired, I have found, I need scarcely say, of the utmost assistance to me in my inquiry. At this time the disease is to be found in the death registers, which themselves bear many internal evidences of the fact, under the name of its most prominent symptoms, or of its often fatal complications; and it could scarcely be otherwise. The disease did not, as I have said, correspond with any to be found described in the British medical literature; owing to its rapid course when once urgent symptoms set in, the medical practitioner had often, even usually, only an opportunity of seeing a case shortly before death, when very probably some localization in a vital organ had

masked the fundamental morbid process; and, lastly, the cutaneous lesion, which might of course have formed so important a diagnostic sign, was either not observed in these early cases at all, or its significance was not recognized. But in 1878, Dr. Bell, of Bradford, after having an opportunity of watching one or two cases, assured himself that the disease was of the nature of a general blood poisoning; and from that time he advocated this view with much energy and success. The disease came to be called the "Woolsorters' disease", and is occasionally found recorded in the death registers as "pyæmia", "septicæmia", "blood-poisoning", etc. At the time the Government inquiry was ordered it was further suspected that it might be anthrax.

My inquiries, the results of which on this head may be summarized as follows, have placed this latter view, I conceive, beyond question.

1. In the course of the inquiry I had an opportunity of making *post-mortem* examinations in five cases, the clinical features of which had stamped them during life as more or less representative cases of the mysterious disease to which the sorters are subject. In each of these five cases the pathological appearances of anthrax were discovered, anthrax bacilli were found in the blood and other fluids, and in different organs; and in two of the five cases in which the autopsy was made very early after death before the fluids removed showed signs of decomposition, inoculation by Dr. Greenfield of these fluids produced typical anthrax in animals. A sixth case that occurred in my absence from the district was shown also, by the last-named direct experimental method, to be anthrax.

2. In several other cases (nine occurred during the time of, or shortly preceding, my inquiry) diagnosis was placed beyond doubt by the appearance of the typical cutaneous localization of the disease, the malignant pustule. These cases were clearly associated in their causation with other cases of the internal form of the disease.

3. Other cases almost certainly owned the same origin as did unequivocal attacks of anthrax in sheep and cattle. That is to say, the water used in washing certain wool that had, it may be confidently affirmed, infected several sorters just before the date of my inquiry, was run in ordinary course upon a neighbouring field, and animals pastured here, and that had been seen to drink of this refuse water, died of what was proved to be anthrax.

4. Although every effort was made to discover the occurrence of sickness of a similar suspicious character amongst

others than those engaged in the manipulation of wools and hairs, and, among those so engaged, to trace the disease to other possible sources of infection than those associated with the occupation, such efforts were entirely unsuccessful: not even in families of suffering wool-workers, where all were apparently exposed to the same home influences, dietetic and other, were any but those engaged in the occupation attacked; and, again, the disease never seemed to spread from the infected sorter to other individuals. In short, the wool was found to be the medium, and so far as Bradford experience could show, the sole and necessary medium, of this infection. What known contagium would correspond with this experience?—that is to say, what contagium is likely to show this exclusive choice of vehicle in transportation to man? We can only, I think, in reply point to anthrax.*

5th and lastly, there is evidence that amongst the Turkish goats, whose fleeces seem to be the most productive of this mischief amongst the sorters, there often prevails a disease that corresponds in the accounts of its symptoms with anthrax; and men in Constantinople who have the handling of these fleeces before exportation, are likewise subject to a disease, characterized by the appearance of cutaneous pustules, and similar in other respects to that from which the English sorter suffers.

Pathology.

Symptomatology.—Anthrax is usually divided into two principal varieties; that presenting a characteristic malignant pustule, and that having no characteristic external lesion—the so-called internal anthrax. The two forms are etiologically identical, but in a clinical view the distinction is a convenient one.

I will pass briefly over the external form, for the malignant pustule is described in every text-book on surgery, only stopping to note the varieties under which it may apparently be developed. Of these varieties four may be distinguished:—(*a*) The typical malignant pustule, forming apparently at the point of inoculation of the poison, and remaining throughout a purely local process; (*b*) the same lesion accompanied

* The occasional presence of decomposing matters, dung, blood, pieces of flesh, etc., amongst the fleeces, has been much remarked upon as a possible cause of blood poisoning amongst the sorters. My experience leads me to believe that the frequency of such impurities has been considerably overstated. Moreover, we have no reason to believe that the products of ordinary decomposition of substances of this kind will give rise to an acute idiopathic septicæmia, supposing the disease under consideration were of this nature only.

or followed by constitutional symptoms; (*c*) the same lesion, but formed by secondary localization of a constitutional infection; (*d*) a minor pustule or small slightly inflamed tumour or boil.

The latter has not hitherto, I believe, been ascribed to the specific infection; and its appearance is very different to that of the typical malignant pustule. It has no central black eschar, nor raised vesiculated border. It commences as a papule, develops into a small tumour, or slightly inflamed eminence, with a raised central point, less prominent perhaps than in the ordinary furuncle. It terminates in resolution or exudes only serosity; or, under the influence of irritation, deep suppuration may occur. While the swelling itself gives rise to comparatively little pain, or even increased sensibility, there is often a tendency to a diffuse cellulitis. Abatement is followed by exfoliation of the cuticle, usually for some distance around, and by some local anæsthesia. I did not happen to see this local lesion in woolsorters in the stage in which microscopical observation would be likely to be of value; but there is no doubt of the fact that, when sorting certain wools, the men frequently suffer from it. Medical practitioners in making *post-mortem* examinations in cases of woolsorters' disease, are apt to suffer in like manner; as also are men engaged in slaughtering or dressing cattle affected by splenic fever. It would seem indeed, from the evidence, inevitable to regard this lesion as owning the same nature as the more pronounced "malignant pustule"; differing in its manifestation from the latter according to a variety of circumstances, foremost, perhaps, amongst which are the peculiarities of species and of individuals, and the precise situation on the body at which the introduction of the poison occurs. For in this opinion, as regards the varied results of cutaneous inoculation in the human species, I find myself in accord with M. Colin in his observations as regards the carnivora (*Bulletin de l'Académie de Médecine*, No. 27, 1880). He found that in dogs much the same variety of local lesions, as above described, could be produced; the difference in the local manifestation, and with it the greater or less risk of secondary constitutional infection, appearing to depend largely upon the region of the body chosen for the inoculation of the poison. All varieties, however, of pustule, M. Colin says, are virulent, and are capable of producing, although with different degrees of probability, general and fatal infection. If this be so the practical importance of a careful study of this lesion is obvious.

Constitutional symptoms, when developed in these cases of

malignant pustule, are of the same nature as those I am about to describe under the head of "anthrax fever".

The course of the so-called "internal anthrax", or as I would prefer to call it "anthrax fever", is divisible, like that of other infectious diseases, into stages. There are, the stage of incubation; the stage of invasion or prodromata, in this disease one apparently of very variable duration, and marked sometimes by intermissions. Then follow, if the infective process progress, suddenly, the acute and stormy toxic manifestations that characterize the fully-developed disease. These latter symptoms, when developed in such degree as to keep the patient imperatively to his bed, terminate usually; but not always, in death: and this is usually in from two to five days from the commencement of such symptoms. It is possible that this stage, thus developed, cannot truly last much longer, although relapses may apparently occur; seven days is the limit that Dr. Bell of Bradford, who has seen many of these cases, places upon it; this, too, is the time, at about the sixth or seventh day after definite commencement of acute symptoms, that improvement, when recovery is to take place, commences. But although he pass this period the patient may succumb later to secondary inflammatory processes—processes in which the lungs are specially apt to be implicated. In cases thus progressing, a scientific diagnosis, if one have not already been arrived at, will often (as may readily be imagined) be almost impossible.

The Period of Incubation.—In the external form of the disease previous observers have remarked upon the variableness of this period, I have estimated it to extend from a few hours to twelve or fourteen days. The Bradford experience tends in the main to corroborate this estimate for the constitutional infection; but it seems to me to go further, and points very distinctly, I think, to the possibility of at times long periods of latency of disease germs within the system, to be followed by development only when favourable conditions arise. Certain cases, in which the sufferers had for some weeks discontinued working on what has hitherto been looked upon as dangerous material before their attack, suggest this possibility; and still more forcibly, it seems to me, do certain phenomena of the disease that I shall presently have to remark upon, including the occasional prolongation and intermittency of prodromata.*

* In one case the sufferer died of all the symptoms of the woolsorters' disease, his death being certified "congestion of the lungs, two days", six weeks after taking to house-painting as an employment. Previous to that six weeks he had been sorting a quantity of alpaca that was known to have

Prodromal Stage.—The sufferer usually first complains of chilliness, not often amounting to distinct rigors, bodily weariness often very great, and mental depression. There are usually more or less profuse perspirations, and irregular flushings of the face, which may appear congested or pallid, usually the former; sleep is restless, or there is sleeplessness. The respiratory centres are commonly early involved in this nervous disturbance; there is a sense of constriction of the chest, accompanied by shortness of breath, frequent sighing and yawning, and later, perhaps, by cough. Aching of the limbs is usually complained of, perhaps cramps in the calves; and there may be peculiar feelings of numbness or formication in the extremities; or less commonly loss of power and staggering gait. Not quite so commonly perhaps as some of these other symptoms, but still frequently, there is headache, with slight pain or stiffness in the neck, dizziness, nausea, and vomiting; and vomiting may, indeed, be the initial symptom.

Should the disease progress, in a variable time, sometimes in a few hours, more commonly perhaps in from two to five or six days, these symptoms, especially the sense of weariness and prostration, increase, or others more alarming present themselves, so that the patient finds himself incapacitated from work. In other cases it seems that distinct intermissions occur; and if we could be sure that in the histories of such attacks the first symptoms were rightly ascribed to the specific cause, and if we could eliminate the possibility of a fresh or intermediate infection, it would appear that weeks might intervene with intermittent prodromata before the stage of acute symptoms was reached. The frequency of distinct remissions at this time is unquestionable; periods, too, of excitement may alternate with those of lassitude and weariness. Thus it not unfrequently happens that when a man has given up work and taken to bed feeling utterly prostrate, he will think he can, and sometimes does, return; and he may thus work again for a few hours before his final discontinuance of active exertion.

The Stage of full Development.—Although the stage of in-

infected other men. Another man had been sorting clean English wool for six weeks before his fatal attack; before that he had been engaged along with other men, who likewise suffered from fatal attacks of the disease in sorting Van (Turkey) mohair. In other cases there seems to have been, at first, an abortive infection, then a period of absence of all symptoms, and then in a variable time a second, sometimes fatal, outbreak. The possibility of the latency of the poison had been forced upon the sorters themselves from the facts they had collected as to the behaviour of the disease, and when first I went to the district, I was asked by a deputation whether such a condition was possible.

vasion may have been prolonged, that of full development is usually sudden and unexpected. The prostration and restlessness become more marked; the face may be deeply flushed or pallid; the respirations are almost always greatly accelerated, to 30 or 40 perhaps, while the pulse rises to 100 to 130 or 140 per minute, and is usually weak and compressible. The temperature, although further observations are needed, often shows no considerable rise above the normal, and is moreover very variable, not only in different individuals, but irregularly so in the same patient. At this early period, twelve to twenty-four or forty-eight hours after appearance of urgent symptoms, whilst normal respiration may still be heard, but with increasing precordial anxiety and dyspnœa, with signs of less, and less complete filling of arteries, with coldness of surface, and deep cyanotic hue of skin, the patient may sink into a state of fatal collapse. Or, apparently, a similar condition, or perhaps one of asphyxia, may be induced at this time quite unexpectedly, with a previous temperature only moderately high, and a cardiac weakness not excessive, from the rapid pouring out of a vast serous effusion into the pleural cavities. But the case is usually a little more prolonged; some reaction takes place, and the symptoms above enumerated are supplemented or accompanied throughout by others. There may be more marked and continued fever, with a temperature rising to 104 deg. or 105 deg.; but always accompanied seemingly by a tendency to irregular downfalls, and the breaking out of more or less profuse perspirations. The symptoms of the prodromal stage are all increased; violent headache, incessant vomiting, increased restlessness and excitement, vigilance, delirium or somnolence; or, perhaps more frequently, the sufferer retains his mental faculties, only comparatively impaired, until the end. Often quite early in the disease symptoms of various acute local congestions arise. The lungs are specially apt to be affected; and the physical signs of that condition, with the occurrence of a copious frothy blood-stained expectoration, are noted; and a fatal œdema may supervene; or more copious hæmoptysis, as might be expected from the pathological conditions, may occur. Similarly, more or less severe gastro-intestinal symptoms may appear. From the first there has probably been loss of appetite, with a moist furred tongue, afterwards, perhaps, becoming brown and dry; now colicky pains may trouble the patient, with vomiting (probably of different significance to that of the initial stage), and diarrhœa, and sometimes jaundice. Hæmorrhage from the bowels has not been observed

in the cases on which this description is founded; but it has been noticed by different observers, and one would expect it occasionally to occur.

Sometimes there may occur a remission of all the more distressing symptoms; the breathing becomes easier; the colicky pains and vomiting cease; the restlessness and sense of oppression diminish, and a little sleep even may be obtained; the patient and his friends believe, even as the end is approaching, that recovery is close at hand. But the profound impression that has been made upon the circulatory system soon manifests itself anew. Perhaps it is suddenly discovered that the extremities are stone cold; or syncope, lasting only a few minutes, may suddenly occur; cyanosis, before noticeable at the lips and finger-nails, gradually spreads and deepens; the evidences of cerebral mischief may now become more direct and violent, with spasmodic twitching of the brow, wild delirium, and convulsions; or a group of symptoms referable, perhaps, to the implication of the spinal cord in the prevailing morbid changes may be evoked; two cases of tetanic spasms, one commencing with trismus, were described to me; or the patient sinks into profound coma, it may be, as shown in one case, from cerebral hæmorrhage, from which there is no arousing; or the stages of a typical asphyxia close the scene.

Should the patient survive this stage of full development he may still, as already stated, succumb later to inflammatory processes, set up during its continuance; these processes would appear to be frequently of embolic or thrombotic origin, and they correspond closely with similar changes one meets with in pyæmia.

But recovery after this stage, I am led by this inquiry to believe, is not by any means so rare as hitherto supposed. Besides the rudimentary and chronic cases, presently to be spoken of, several, running more or less the course I have described, were related to me, where complete recovery supervened. The convalescence will be long and tedious; a tremulous and feeble condition of the muscular system is left; the cutaneous sensations noticed in the prodromal stage (numbness, "furry" feeling) will very likely continue for a time; muscular cramps may continue to trouble the patient; the epidermis sometimes desquamated, in two cases the finger-nails came off; various eruptions are apt to appear; herpes, boils, and sometimes, it would seem, gangrene. And these latter manifestations may occur during the acute stage.

And besides these developed cases there are—the evidence,

I think, admits of no doubt—many cases of internal anthrax of a milder and rudimentary form. Often it happens that while one man will suffer from the marked and rapidly fatal malady, his comrade, working on the same material, will sicken in a precisely similar way. But, after various duration of the symptoms, at a point short of the intenser manifestations of the disease, evidences of improvement appear. The patient, according to the severity and duration of his attack, quickly returns to work, or passes through a long and tedious convalescence. Further, the histories of long-continued *malaise* are too numerous to ignore, and they also are associated with unequivocal and fatal attacks in others. The symptoms in this class of cases are much like those of the prodromal stage of the acute infection, but may continue for an indefinite time, and often manifest a peculiar periodicity, the men frequently thinking that each time they have opened a bale of wool they have been affected afresh. These manifestations culminate occasionally in an acute attack of the disease (in which case, they may be regarded as prodromal symptoms); but this is by no means constant, and often they disappear spontaneously. It may be that there is a condition of chronic anthracoid intoxication, the analogy of which would be found in malarial disease.*

Permit me to diverge here for a moment from the main subject to speak of the apparently close pathological affinities between these two diseases, anthrax and malarial fever; affinities and similitudes which gain new importance and significance in the light of the recent observations of Klebs and Tommasi, by which it would appear extremely probable that the virus or contagium of malarial fever is a vegetable organism of the same species as the bacillus anthracis. I have already remarked upon the etiological relationship between these affections. The occasional long latency of the disease germs within the system that apparently characterises both, constitutes a fundamental distinction between these two

* Here is an example of the numerous histories of this kind obtainable. The sufferer had been sorting "Cape mohair", and one of his fellow-workmen, employed on the same material, died at the time from anthrax. He writes me: "My symptoms were headache at times, aching limbs, cramps in my fingers, thighs, and calves (often had to jump out of bed at nights with cramps in my legs). very sore and raw nostrils, breathing through the nostrils hot and painful, a dull aching pain across the eyes, breathing sometimes quick and difficult, the least exertion thoroughly exhausting me. After meals heavy and very sleepy. No sound sleep at night, vomiting occasionally after meals, and first thing in the morning. I was obliged through circumstances to stick to my work as long as I possibly could, did not stay away more than a day and half a day at a time, till obliged to leave it."

diseases on the one hand, and most of the other specific infections, although not all, on the other. So marked is this phenomenon in the case of malarial fever, that in the history of many attacks the malarial poison would appear almost to rank as a predisposing rather than as an exciting cause, a factor only capable of acting upon the interposition of some additional accidental influence, extrinsic to the infected individual. And in the case of anthrax, how otherwise than by aid of this doctrine of latency can the occurrence of prolonged and intermittent prodromata be accounted for, to say nothing of the few cases in which the first symptoms have followed only at a considerable interval after known exposure to infection? Moreover, I shall presently bring forward evidence which suggests that other tangible influences, extrinsic to the individual, besides the initial exposure to infection, may be operative (and if perceptibly operative, then, presumably, in some degree, necessary) in determining the full development of the disease. Of very similar significance to this condition of latency of the poison is the almost established occurrence, likewise common to both diseases, and likewise unassociated with most other specific infections, of a condition of chronic, as well as of acute, infection, as well as the important fact (that seems, from histories which I have detailed in my official report, to be well ascertained) that one attack of the disease successfully overcome confers no immunity from subsequent infection. The symptoms, again, of the acute form, together with the manifold localisations of the poison in such cases, and the anatomy also of this form, follow closely the descriptions accorded to pernicious types of malarial fever, as seen in tropical regions; and although the regular periodicity of typical attacks of intermittent fever has never, so far as I know, been observed, yet frequent remissions, and even complete, although irregular, intermissions, constitute a marked feature in anthrax fever. Further, in the descriptions of what I have designated the chronic form of the disease, one can scarcely fail to be reminded of the so-called malarial cachexia of malarious regions. Bechi, for example, says that conditions, that will produce in such districts attacks of fever in susceptible individuals, lead to an indescribable unrest, lassitude, and muscular debility in many strong robust men. All the other chronic symptoms, too, so often complained of by the sorters of foreign wools—dizziness, disposition to vomit, headache, dry and sore nostrils and mouth, oppression of chest and hurried respirations, perspirations, with an apparent periodicity and similar sequence of these symptoms, and an occasional appearance of

petechiæ upon the skin, and herpetic and other eruptions, are all found in this malarial cachexia (or, as it is termed in Holland, "burnen koorts", or inward fever). There is obviously an analogous constitutional disturbance.

Pathological Anatomy.—Evidences of early decomposition are commonly met with, parts of the body being considerably swollen and discoloured, especially around the neck and about the chest and arms, where, also, there is frequently an emphysematous condition of the subcutaneous tissues. Besides these *post-mortem* appearances, various pathological conditions of the skin may be noticed; petechiæ being apparently the most common. The heart is often flabby, and the muscular substance dark in colour, and containing sometimes minute hæmorrhagic extravasations; the blood in its cavities and in the large vessels is generally quite fluid, and of a dark cherry colour, and the lining membrane, unless the autopsy be made very early after death, usually shows a corresponding tint. There is often a considerable pericardial effusion, and both pleural cavities may be found filled with a straw-coloured serous fluid. The lungs may be normal, or more usually they exhibit signs of considerable hyperæmia, combined, it may be, with œdema. This hyperæmic condition may, it seems probable, go on to a true pneumonia; or metastatic pneumonic processes may be set up, with the formation of numerous small infarcts and abscesses. The lungs, too, like other organs, may exhibit numerous hæmorrhagic infarctions. The bronchial glands are very usually swollen, soft, and of a purple or blackish colour. The bronchial mucous membrane may or may not be reddened, small hæmorrhagic extravasations may frequently be seen beneath its substance, and in the bronchi blood-stained mucus is sometimes found. The mucous membrane of the pharynx and trachea is commonly hyperæmic. In the abdominal organs, also, signs of congestion, with various hæmorrhages, are usually met with. The spleen is usually, although not constantly, enlarged, its substance soft and diffluent; the liver is less frequently hyperæmic, may indeed appear anæmic; the kidneys are usually much congested, and show signs of cloudy swelling. The mucous membrane of the intestinal tract will occasionally exhibit signs of acute catarrh, and the mesenteric glands will probably then be congested and swollen; but this condition is not constant, nor have I been able to find the intestinal anthrax carbuncles spoken of by some authors. The bladder in two of the five autopsies conducted by myself, was quite empty, in two the urine contained albumen. The brain may exhibit no con-

spicuous lesion, or there may be extensive hæmorrhages into its substance or in the membranes. Perhaps one of the most constant and striking lesions are the serous, or sero-hæmorrhagic jelly-like infiltrations in various connective tissues. They abound sometimes in all the loose connective tissues of the body; especially in those of the neck, in the mediastinum, in the sub-pleural cellular tissue, and in the loose connective tissue around the kidneys. They give the organs and vessels in the neck the appearance of being embedded in and surrounded by a jelly-like plasma, and the lungs of having jelly-like masses appended to them; they may be of a yellow or straw colour, or of various blood-tints; or the blood sometimes appears to have been extravasated over an already gelatinised effusion. Lastly, the specific contagium of the disease, the bacillus anthracis, will usually, in my small experience always, be found, although in widely varying quantity, in the blood and extra-vascular fluids.

Modus Operandi of the Poison.—If a clear view could be obtained of the mode of action of this contagium upon the system, and of the nature of the phenomena to which its operation there gives rise, a far more useful purpose would obviously be secured than by any mere enumeration of individual symptoms,—varied as they are and difficult to describe. Two principal theories have in this regard been advanced: one by Bollinger, who attempts to show that in the rapidly fatal attacks the fungi develop in enormous number in the blood, absorb (as this bacillus and others of the same group have been shown by Cohn and others to do) the oxygen of the red-blood corpuscles, and in effect that they kill the organism attacked by carbonic acid poisoning; and he explains the fever and other symptoms evoked in cases of a slower course by the conjecture (a conjecture in some degree justified by the well-known researches of Pasteur) that other chemical poisons are secondarily produced in the blood by the agency of the bacillus. Toussaint, on the other hand, maintains that to the mechanical action of the bacilli, in obstructing the circulation, by their impaction in the capillaries, the manifestations of the disease are mainly attributable. Both these processes, it can scarcely be doubted eventually if the disease progress, play an important part in the production of the morbid phenomena. But against both it may be urged that the bacilli are rarely found in large number in the blood of man before death; more usually, perhaps, we do not succeed in finding them at all at this time. Moreover, the symptoms of carbonic acid poisoning are not the early prominent symptoms of the

disease; in some respects, indeed, appearances would seem to point clearly to this, and to the diminished supply of oxygen, as quite secondary toxic manifestations. For example, the heart's action appears always to be accelerated. If the cardio-inhibitory centre be acted upon at all, it is apparently paralysed, not excited, as we believe it to be in carbonic acid poisoning. It is true, bacilli may exist in large number in various organs before they can be found in the general circulation, and if it could be shown that they early caused mechanical obstruction in the cerebral capillaries, as indeed from the narrowness of these capillaries we might expect them to do, it might reasonably be conjectured that by the cutting off of the blood-supply to certain nervous centres, by the establishment of collateral hyperæmia, or of, by reason of the septic character of the emboli, inflammatory changes, and so on, such disturbances might be produced as would result in the nervous phenomena that characterise the accession and progress of the disease.*

But all that can be said with safety at present is that the virus of the disease produces by some means a profound functional disturbance of different nerve centres. All the symptoms of the prodromal stage may be attributed to such influence. Thus we have the rapid increase of cardiac and respiratory movements without structural lung mischief, and without, often, the increase of temperature on which such accelerated movements in fevers perhaps ordinarily depend. There is the early prostration, the dizziness, the profuse perspirations, the restless sleep or insomnia, and the subjective cutaneous nervous symptoms. The nausea and vomiting are probably also attributable to central nervous influence; for the vomiting may suddenly cease, to give place to other symptoms. Especially, it seems to me probable, are the vasomotor and trophic nerve centres affected. We have the early disturbances in the circulatory system, the early prostration, and cramps attributable also probably to muscular mal-nutrition; following, we have speedily developed local hyperæmias and venous congestions, and various evidences of acute

* Wagner found bacilli early crowding up the cerebral vessels, although this, it must be said, was not found to be the case by Koch. It has been thought that in certain malarial diseases pigment embolisms of the cerebral capillaries, affecting considerable territories, may originate the nervous symptoms; and the new light that has been thrown upon the pathology of this disease by the isolation of its particulate contagium, may possibly lead to an elaboration of this theory. Bastian has suggested a relation between capillary occlusion and the cerebral symptoms of fevers in general.

nutritive disturbances. We can scarcely believe, for example, that capillary thromboses and embolisms alone will cause the extensive œdematous and hæmorrhagic effusions often found in almost all the loose connective tissues and elsewhere in the body, and which occasionally give early evidence of their presence during life; but if we suppose, and it seems to me we have every reason, an acute disturbance of the nutrition of the walls of vessels, and of the tissues through which they pass, through some central trophic influence, combined with diminished tonicity, and consequent local congestions, from vaso-motor disturbances, an unusually fluid condition of the blood unusually charged with carbonate acid, and a weakened heart, such effusions are what we might expect to ensue. Then, again, similarly explicable, without any marked or continued hyperæmia of the skin, or great heat of surface, there is often in cases of recovery desquamation of the epidermis, and sometimes falling-off of the finger-nails; and further, pointing in the same direction, there is the long and tedious convalescence marked by great bodily weakness. It might be thought that other of the cutaneous lesions, the petechiæ, the herpetic eruptions, the pustules, and the local gangrenes, that often mark the convalescence, or occur during the progress of the attack, may owe their origin to a similar trophic disturbance. We know how herpetic eruptions occur when certain nerves are irritated (in herpes zoster, cerebro-spinal meningitis, etc.), how pustules appear after the injury or experimental division of certain nerves, how speedily gangrene supervenes upon certain injuries to the nerve-centres. More, however, could probably be said for the theory of an embolic origin of these cutaneous lesions. The histology of the disease teaches us that occlusion of the capillaries often actually occurs, and on the other hand it has been pretty fairly shown that such occlusion of the cutaneous vessels may give rise to corresponding lesions; for example, to the petechiæ in diphtheritic endocarditis, to the petechiæ and pustular eruption in malarial diseases, and in septicæmia and glanders; and possibly in small-pox, as Zülzer contends, the enormous number of micrococci found in the cutaneous vessels in that disease may have a causative relation to the pustular eruption.

To the subject of individual susceptibility I would ask the remainder of your attention. This feature, a marked one in nearly all infections, is, as I have shown, peculiarly marked in the various forms of anthrax; and such is the case also with the disease as it is found in the lower animals.

It has long been known that whereas herbivorous

animals frequently suffer from this disease in an epizootic form, and are most readily inoculated with the poison, omnivorous, and to a still greater degree carnivorous, animals are with difficulty infected. The blood of the carnivorous race is perhaps deficient of something which is necessary for the free development of the poison; or contains something in greater degree inimical to its life. Experiments by M. Colin (quoted by Bollinger), go to show that the gastric juice of the dog is destructive to the poison. The observations of Professor Feser, of Munich, however, recently published, go further, and seem to promise a practical issue. He had observed that sometimes the rats he was experimenting upon exhibited a marked immunity from the effects of inoculation, and by a long series of experiments he assured himself that their susceptibility or immunity was only due to their alimentation. Those fed on flesh could not be inoculated; those fed on bread-crumbs or grain were inoculated with ease.

Here is one of his series of experiments. A female rat, previously unsuccessfully inoculated, having been fed on flesh only, brought forth nine young ones. The family was fed on thirty grammes of anthrax meat, and again the same with bread steeped in anthrax blood. No harm ensued. But the mother's diet was changed to bread. She was then fed on the flesh of a rabbit that had suffered from anthrax for two days. She was found dead in her cage three days later. Of the nine young ones, three were placed with three of the progeny of another mother, and fed only on cooked beef and horse flesh, while the remaining six were fed on bread. All of them were inoculated with the same quantity of anthrax blood from the heart of a rabbit; all those fed on bread died rapidly between the twenty-second and thirty-second hours; the six fed on flesh remained perfectly healthy.

From the first I was unable to dissociate these observations from a practical bearing upon the Bradford inquiry; and I consequently set to work to ascertain minutely the alimentation of the sufferers from this disease. But before giving the results of this investigation, I may refer to a letter bearing upon the subject, that reached me, in the midst of my own inquiries, from a wholly independent and unexpected source from Constantinople. Anthrax in Constantinople appears to be well known, and it engages the special attention of a certain class of practitioners, called "dallack doctors", "dallack" being there the name given to the disease. One of these, a M. Diamanthi, enjoys a great reputation, "he only pretends to cure dallack" and his skill in this "has been handed down from father to son for several generations." The treatment

he adopts (which appears to refer only to the malignant pustule, and to have nothing special in it) is described; and "the diet to be observed is caviare and bread, and a little spirit, vegetables and fruit being especially dangerous". Thus it would seem that the "dallack doctor" has arrived at a conclusion pointing distinctly in the same direction as that to which the scientific experiments above referred to lead. I, on the other hand, have to relate some very curious and suspicious circumstances that may admit of a similar interpretation; and which, perhaps, will be so interpreted when further opportunity for research has been made use of.

Early in the inquiry the fact attracted attention, that in an extraordinarily large proportion of cases, the initial or prodromal stage of the disease may be said to have terminated, and the acute, often fatal one, commenced between the Saturday evening and the Monday morning. Thus of twenty-nine recent cases of acute internal infection that I have taken as affording more exact data, in twenty-one the sufferers practically left work for the last time at the completion of the week's labour on the Saturday afternoon. I say practically, for two of these twenty-one attempted, but unsuccessfully, to resume work on the Monday, and one, instead of going to his work on the Saturday, went for a long walk, thinking to throw off his feeling of malaise. Moreover, of the eight remaining cases, three left work or took to their beds during special holidays, viz., one on New Year's day, one on Good Friday, and one on Whit-Tuesday. So that in 80 per cent. of recent cases, the acute stage may be said to have commenced on a holiday. This coincidence is not, moreover, it seems, a new feature. It had been noticed in earlier attacks, so that I am told it was often said amongst the men, that if a sufferer got over the Sunday (that is, the Sunday following the attack) he was all right.

I could not, on the most careful inquiry, attribute this incidence of the disease to any peculiarity of the occupation. There is no special work confined to any one day of the week. The work-rooms are usually swept out on the Saturday, so also are they in most factories on the Wednesday, and sometimes every day; and owing to a plentiful sprinkling of water there is certainly less dust at such times than when the bales are opened; the latter operation being, so far as time is concerned, indifferently performed. The explanation that of course next suggests itself is that the prodromal stage is long and indefinite, and that the men struggle on with their work to the week's end, thinking that the day and a half's rest they will then have (Saturday being a half holiday) will be a

sufficient restorative. But in examination, this explanation, likewise, does not appear to me satisfactory. There is very little evidence of this struggling on and gradual breaking down.

Ten of the twenty-one were not known to have complained of sickness before the Saturday of their leaving work; others seem to have felt no worse on that day than on preceding ones; and they, one and all, appear to have gone after dinner for their usual Saturday afternoon's walk, or other recreation. Still, ten of them (the evidence was doubtful in one case) had complained more or less before, and some few had very probably to make an effort to complete the week's work. But granting that they have in this way the termination of the prodromal stage, as indicated by the cessation of work, to an extent in their own hands, so to speak, it does not follow that this would determine the accession of the stage of full development; and yet, at some time after leaving work on Saturday afternoon and before the Monday morning, this stage, with urgent symptoms, had set in. This peculiar feature of the attacks may be expressed as follows:—

Of the twenty-one men giving up active employment on the Saturday, two were seriously ill the same night, eighteen others were similarly placed by the Sunday night, so that on the evening of the last-named day, of these twenty men one and all were confined to bed through sickness, although, as before stated, two attempted to resume work on the following day. In the case of the single remaining sufferer the attack was a little longer delayed; he had left work apparently quite well on the Saturday, had gone on a holiday excursion to Kidderminster, and was brought home in a partial state of collapse on the Tuesday night. The day of termination will show, again, how swift in these cases was the progress of the disease after the Sunday. Of the twenty-one cases, five recovered; all but one of the remaining sixteen had terminated fatally by the following Friday night.

The suggestion is forced upon the mind, by an attentive study of these histories, that between the hour of leaving work on the Saturday afternoon and the Sunday night something must occur to awaken as it were the slumbering disease germs, or to give an enormous impetus to the disease.

There is one marked difference between the mode of living of the large majority of these workers on the Saturday and Sunday, and on the other days of the week. They all, it seems, on the work days make more or less hurried meals; a large number, indeed, (in some factories two-thirds of the

men) take their meals to their work. It follows, that on Saturday, when they dine at home, but more especially on Sunday (on this point testimony is unanimous), a much larger amount of vegetable food is eaten than on other days; as much often, I am told, as on all the other days combined. Further, during what is afterwards assumed to be the initial stage of the disease, a man usually thinks he is suffering from a cold; and it is a very common practice indeed for him to lay up on the Saturday evening and Sunday, when he would not on other days, and doctor himself. With this latter object, for the purpose of "sweating himself", he drinks copious draughts of various herb decoctions. In twenty-two recent cases of the disease, where special inquiry was made on this point (attention was not directed to it at the early stages of the inquiry), the practice of drinking such infusions had been carried out, during the stage of indefinite initial symptoms, which, in many cases, it will be remembered, seem to prolong themselves or to disappear spontaneously, in no less than fifteen; and whether there exist a causal relation or not, it was not very long after this imbibition that the violent symptoms of the disease manifested themselves. In nearly every case where satisfactory information was obtainable, and it was obtained in the large majority, the development of these violent symptoms was found to be co-incident with the ingestion of an apparently unusual quantity of vegetable food in some form or other (onions, lettuce, cabbage, fruit, or herb decoctions). In the progress of certain of the cases, again, after remission of the symptoms, a relapse or exacerbation appeared to follow the ingestion of vegetable food or fruit. (This was particularly observed in three cases; the patients were much better, and thought to be recovering, and were downstairs eating fruit, and in one case cabbage also, twenty-four hours before the fatal termination of their illnesses.) At the same time I must guard against an appearance of laying undue stress upon this coincidence. I am fully alive to the danger of drawing deductions from the mere concurrence of two circumstances, when one of them is so common a one as the eating of vegetable food and fruit. Still, the evidence above given is strong; and, if the interpretation I have suggested for it be correct, is of infinite importance.

It is conceivable that certain alimentary substances may bring about in the body such chemical or morphological change as will render its fluids a richer field for the proliferation of disease germs.

THE TRANSMISSIBILITY OF DIPHTHERIA FROM ANIMALS TO MANKIND.

By GEORGE FLEMING, F.R.C.V.S., Army Veterinary Inspector, War Office.

[*Read: May 31st*, 1881.]

THE transmissibility of certain diseases of animals to our own species is a subject of great, and even of supreme interest and importance, when we consider that the majority of them are maladies of the most serious and fatal character. It is somewhat strange that while man is capable of receiving a number of disorders from different animal species, these receive scarcely any from him; so that the danger of transmission is nearly all on one side.

When I mention rabies, anthrax, glanders and farcy, variola, probably a form of enteric fever, in all likelihood tuberculosis, and very possibly diphtheria — to say nothing of several parasitic disorders due to animal or vegetable organisms—it will be seen that the list of diseases which may be conveyed from animals to man is a formidable one both in quantity and quality. And what renders the subject still more interesting, is the fact that from time to time another disease is added to the list of scourges we receive from our co-denizens.

Persons who mix much amongst animals are, of course, most exposed to disease from them; but, nevertheless, they who may have no communication with them at all are not exempt, as the flesh or milk they consume, or the articles or materials of animal origin which they handle, are vehicles by which they may become infected.

Looking to the discoveries made in the past, it is not at all unlikely that we shall find that other diseases, whose origin is as yet unknown, or unsuspected even, are derived in some way or other from animals. Tuberculosis is the last disease added to the list of those which may be transmitted—judging from analogy and a few pathological facts—to the human species; and we have now to discuss whether diphtheria may not also be transferred from animals to man, and ,if so, by what channel or medium.

The serious nature of this disease, its fatality, its alarming complications and troublesome *sequelæ*, and its appearance oftentimes in an epidemic or epizoötic form, render it worthy of the closest study, and particularly with reference to its origin and prevention.

Diphtheria, or diphtheritis, as is now well known, is not only a very contagious and infectious disease, but also—thanks to the very careful investigations and experiments of Hüter and Trendelenburg, Nasiloff, Oertel, Eberth, and more recently of Talamon—inoculable, and due in all probability to a special parasitic vegetable organism, which can be cultivated artificially. This organism has been discovered in the blood, urine, and other excretions and secretions, as well as in the morbid products of the malady, but particularly in the false membranes which form on the surface of mucous membranes, and constitute a very marked and characteristic feature of the affection.

Of the parasitic nature of diphtheria there can now scarcely exist a doubt. M. Talamon, one of the latest investigators, has succeeded in cultivating the microscopical organisms from diphtheritic matter derived from eight cases occurring in mankind. In the condition of complete development they presented a characteristic mycelium and spores. The former are tubes with partitions, at intervals, from two to five thousandths of a millimètre in length. These, under favourable circumstances, elongate and bifurcate, the bifurcations being characteristic in consequence of their incurved branches, like the sides of a lyre. In other conditions the mycelia do not become elongated, although they multiply so as rapidly to cover the surface of the cultivation liquid; they remain short and assume irregular forms, and give rise to numerous straight rods. The spores are of two kinds, round or oval, which may be termed the spores of germination, and rectangular spores or conidia. The latter characterise the species. They form small rectangles of various sizes, their length being sometimes fifteen thousandths of a millimètre. They may be isolated or united in festoons or zigzag chains. At first homogeneous, they soon become filled with small round granules, highly refracting, and of the size of ordinary micrococci. The round or oval spores are those which, by their elongation, constitute the mycelium. They appear as clear points, from three to five thousandths of a millimètre in diameter, in the middle of a mass of granular material. M. Talamon has inoculated this organism into the mucous membrane of the mouth or nose, or has given it with the food, in six rabbits, two guinea-pigs, four frogs, a fowl, and four

pigeons. The rabbits died at the end of six, eight, ten, and eighteen days. That which died soonest presented an enormous swelling of the neck, resembling the œdema of the neck in diphtheria, and due to a serous infiltration of the connective tissue, and the culture of this serosity showed the same organism with the characteristic conidia. The rabbit which died at the end of eighteen days presented a bilateral fibrinous pleurisy, with effusion. The effusion and pleuritic membrane also yielded, on culture, the organism which had been inoculated; and it could be recognised either by simple microscopical examination or by culture, in most cases, in the liquid contained in the pericardial and peritoneal cavities, and often in the kidneys. The organism was never obtained by cultivation of the blood contained in the heart. The liquid either remained clear, or presented only ordinary bacteria. In the four pigeons, M. Talamon succeeded in producing characteristic false membranes. He first scratched the mucous membrane of the mouth with a bistoury, and then placed some of the cultivated organism in the interior of the mouth. At the end of twenty-four hours a thick membrane lined both sides of the mouth, the tongue, the false palate, and the back of the pharynx. It was yellowish-white in colour, and was formed like the diphtheritic membrane in man, by epithelial cells, fat, micrococci, and bacteria. There were very few of the rectangular conidia, but these were abundantly obtained from the membrane by cultivation. Two of the pigeons died at the end of three days. In one, the entrance to the larynx was covered by false membranes, and the trachea was full of a thick mucus, from which the organism was obtained by cultivation, as also from the pericardial and peritoneal liquids, but not from the blood. The third pigeon remained ill for eight days, and then the false membranes became separated, and the animal recovered. At this point, unfortunately, the experiments have been for the present arrested. They were carried on in the laboratory of the Hôtel Dieu, and some of the physicians, fearing lest some of the organisms might contaminate their patients, stopped the observations.*

Diphtheria has been most carefully studied in man, in whom it is generally localised in the fauces, or pharynx, in the upper air passages, in the spleen and kidneys less frequently; it also involves the nervous system. The peculiar greyish-white membrane or slough which marks the presence of the disease, and is so closely adherent to the mucous membrane beneath, is constantly found in the mouth or pha-

* The *Lancet*, April 9th, 1881, p. 589.

rynx, not beyond; for when the larynx and trachea are involved, the pseudo-membrane formed in them is of a croupous nature, easily detached, and does not usually occasion any loss of tissue when removed.

It is only in recent years that it has been closely studied in animals, though it must be confessed that from the comparative rarity of outbreaks, and the anxiety always manifested to avert loss by stamping it out, much still remains to be accomplished in this study.

The greatest number of outbreaks have been observed in poultry, among which, as well as other kinds of animals, it usually appears in an epizoötic form. Among fowls it is most infectious and fatal. After them, the bovine species appears to suffer most frequently; then sheep, the pig, rabbit, and, finally, the dog and cat—the horse being almost exempt from its invasion, according to some authorities, and as liable as the ox, according to others.

In animals, diphtheritis is, as in man, a general as well as a local disease, and is manifested by acute fever, increased temperature and respiration and pulsation, succeeded by prostration and rapid emaciation, often by sudden collapse: the symptoms varying not only in individuals or species, but in different outbreaks.

In one outbreak, and usually most frequently, the respiratory passages will be chiefly involved — the nostrils, pharynx, larynx, trachea, and bronchial tubes, and even the lungs and pleural membrane being implicated, though the upper air-passages are oftenest the seat of pseudo-membranes. The symptoms in any case are very marked.

In another outbreak the digestive tract will offer the most marked lesions. The buccal, labial, lingual, pharyngeal, and palatine mucous membrane, as well as that of the nostrils and upper air-passages, is swollen, congested, red or violet in colour, and covered in patches by greyish-yellow, tough, fibrinous masses, which are with difficulty removed; though from time to time portions are detached and coughed out, or are carried away by the saliva or nasal discharge, leaving raw surfaces. Deglutition and respiration are performed with difficulty, and the upper part of the neck or throat is swollen and painful. Diphtheritic enteritis is not at all uncommon among animals, and is usually complicated with disease in the mouth and pharynx. Diphtheritic ophthalmia is not an unusual accompaniment, leading to perforation of the cornea, and destruction of the eye; and in bovine animals fibrinous deposits have been observed between the digits and around the pasterns, and the hoofs, and even

the horns, may be shed. In animals suffering from diphtheria, when there happen to be sores or abrasions on the surface of the body, these are very often covered with diphtheritic exudations; while beneath the skin in various regions are sometimes found nodular masses of the same nature as the false membranes. The bladder may be involved; and, in pregnant animals, abortion takes place when the uterine mucous membrane is also the seat of diphtheritic inflammation.

I mention these symptoms and lesions to show that animals are subject to diphtheria, and that it is in them a most formidable malady.

I have said that it is usually observed in an epizoötic form, and that during different outbreaks it will assume special features, particularly with regard to localisation of lesions, symptoms, and especially nervous phenomena, which are at times very distinct. But, generally speaking, the disease most frequently affects the mouth, pharynx, pituitary membrane, larynx, and eyes, and less frequently, perhaps, the thoracic and abdominal organs.

In the majority of cases death sets in rapidly, and is due either to asphyxia, from obstruction in the air-passages, to blood-poisoning (if I may employ the term), or to prostration or sudden collapse. A fatal termination often takes place within two or three days from the first appearance of the symptoms, though in some cases death may not ensue for as many weeks.

An outbreak of diphtheria in animals commences as in other contagious disorders; one animal is first attacked, then another, and so on, until all, or nearly all, have been affected. It is readily transmissible from one animal to another of the same species, and also to other species. In fowls, the extreme facility with which it is spread is largely due, I think, to the healthy fowls picking up with avidity the mucus and shreds of diphtheritic membrane ejected by the diseased. There are many instances recorded in which creatures of other species have been affected from poultry—calves, sheep, and people. In all species young creatures are most predisposed, and this is most marked in the bovine species, the malady being almost entirely confined to animals under a year old.

Of the contagious or infectious nature of the disease in animals there can be no doubt whatever, as there is an abundance of experimental evidence, to say nothing of the recorded cases of accidental transmission, to prove this. Human diphtheria has been experimentally transferred to

rabbits and poultry, and from these to other animals. It has also been accidentally communicated. There is an instance published of a dog which had eaten the fæces of a child affected with the disease. Within a few days the animal died, and, on examination of the carcase, there were found ulcers on the soft palate, and pseudo-membranes on the respiratory mucous membrane.

As has been mentioned, instances are recorded of transmission of diphtheritis from animals to mankind, and this transmissibility is of the greatest moment to us at present. Seeing the terrible fatality attending attacks of the disease in our own species, and the mystery that often surrounds the origin of these outbreaks, nothing should be left undone to discover their real source or sources, with a view to prevention.

Between individuals of the same species the disease is very infectious, and the severity of the attacks and mortality appear to be greater than when it is transferred from one species to another.

The Veterinary Professor Dammann, of Eldena, now Director of the Hanover Veterinary School,* a most trustworthy observer, describes an outbreak of diphtheria which took place among calves in 1876, with transmission to man; and as the relation of the bovine species to our own, so far as food is concerned, is closer than with any other animals, we may glance at the salient points of his narrative.

The veterinary inspector of Ludwigsburg reported that in the course of the previous winter and spring, nearly all the calves of the district, about twenty in number, had died during the first two or three months after birth. At Dammann's request, the body of one of the calves, between two and three weeks old, was sent to him on the day of its death. The appearances in the region of the head, which was most involved, were as follows:—On opening the mouth, the anterior part of the hard palate was observed to be covered with, or transformed into, a greyish-yellow mass. When a section was made of the head, it was found that this mass involved the entire substance of the soft palate, penetrating to the bones, and forming a layer of from one to five centimètres thick. On the right side the mass had made its way into the nasal chamber. Both cheeks were swollen, and when cut into, showed the connective tissue spaces occupied with this fibrinous material; the buccal surface also was covered, but not invaded by it, and much more thickly than

* *Deutsche Zeitschrift für Thiermedicin und Vergleichende Pathologie*, 1877.

the palate. Towards the gum, in the vicinity of the molars, there was a mass about six centimètres in length, and nearly three in thickness, one border of which passed imperceptibly into the substance of the mucous membrane, which was destroyed, and the muscular layer exposed. The mucous membrane of the larynx and trachea was swollen and red, and here and there covered with small purulent masses, but its texture was not destroyed. Both lungs, but especially the right, contained in the anterior lobe a great number of small abscesses, some of whieh projected in a marked manner above the surface, and the interlobular connective tissue in the neighbourhood had become infiltrated with a gelatinous material, and was consequently œdematous. A similar material was observed between and around the toes. Dammann visited Ludwigsburg three days afterwards, and found five calves ill—two in the early stage of the disease, two very advanced, and one on the point of death. The latter had been apparently convalescent a few days previously, but relapsed. The chief symptoms were great dulness and prostration; the animals had no appetite; lay much; there was high fever; frequent short and husky cough; they had great difficulty in standing, the joints being extremely stiff; and saliva flowed from the mouth. There was more or less swelling of one or both cheeks. The inspector himself was suffering from severe sore throat, but his case will be alluded to hereafter.

The disease was evidently contagious, though, notwithstanding that a number of calves had died within a short time, this cause was not suspected.

Dammann had two sick calves sent to Eldena for observation and experiment. He placed them, on May 10th, in a large space in his experiment-stable, so that they could move about. Next to their place of confinement was an aged cow, and he bought a healthy three-days-old calf, and placed it with them. One of the sick calves was submitted to medical treatment; the other, designated No. 1, was left to nature.

On May 11th, No. 1 was very weak, stood with difficulty, drank little, saliva escaped from the mouth, and yellow matter flowed from the nostrils; there was weak cough and diarrhœa. The entire tongue was swollen, and at the posterior part was a large yellow mass. The temperature stood high. The symptoms increased in intensity until the 15th, when the animal was very stiff, swallowing was difficult, and the breathing laboured and stertorous. The fæces contained many micrococci and small rods. The animal died during the night. On examination of the body, early next morning, it was noted that *rigor mortis* was absent.

The blood in the vessels of the head was dark-coloured. The whole of the small intestine was deeply injected, and studded here and there with prominences of a yellow colour, and about the size of peas. The cæcum, colon, and rectum were studded with similar deposits. The mesenteric glands were also enlarged, softened, and greyish-yellow. The right pleural cavity contained about a litre of pus, and the membrane itself was covered with a yellowish-grey fibrinous mass. The pericardium was in a similar condition. The right lung was in places adherent to the side; its right anterior lobe was firm and impervious to air. The posterior lobe, when incised, presented numerous grey and dark-brown patches, varying in size from that of a pea to a hazel-nut. The nasal and tracheal mucous membrane was here and there covered by a purulo-fibrinous mass, though there was no loss of substance. The inner surface of the larynx, on each side, showed two yellow deposits, and on the epiglottis some small roundish yellow masses.

The left side of the tongue, towards the dorsum, and over the entire side, was covered with the same yellowish-grey mass, which involved the mucous membrane, and even the muscular tissue beneath, and was firmly adherent to them. A small deposit of the same character was attached to the hard palate.

The other calf was treated with salicylic acid, and recovered after a serious illness of about a fortnight's duration.

The healthy calf that was put with these two sick calves from Ludwigsburg showed the same symptoms of illness five days afterwards. It remained without treatment for four days. On the second day micrococci and rods were found in the discharge from the nose. On the fifth day the attendant administered a dose of salicylic acid without orders, and continued it until the eighth day, when it was stopped. On the twelfth day the calf showed a swelling on the cheek; this was opened. Recovery took place about the thirtieth day.

Microscopical examination of the deposits on the mucous membrane resulted in the discovery of numerous bright-looking granules. They were scarcely tinted by carmine, nor were they soluble in chloroform, æther, acids, or alkalies. When isolated, they showed decided movements—they were micrococci; there were also rods. These organisms were found in greatest abundance on the superficial portion of the mass, although they were observable in every part; and not only in the pseudo-membranes in the mouth and pharynx, but also in those found in the intestines and about the feet. The result of Dammann's microscopical investigations was to

establish a very close similarity, if not absolute identity, between the diphtheritis of calves and that of mankind. He remarks that the diphtheria of calves is a general as well as a local disease. The lesions are most frequently found in the mouth (cheeks, tongue, and palate) and pharynx; next in the nasal cavities, larynx, trachea, lungs, small intestines, and cleft of digits. In the mouth, the preferential seat appears to be the cheeks, then the tongue and palate.

The principal symptoms are difficulty in standing, stiffness, fever, prostration, and dulness, and the recumbent position, loss of appetite, and difficult deglutition. The temperature is, of course, much increased. There is an absence of thirst, and the diminished appetite may be due to the pain of eating and swallowing. The salivation is most marked, particularly at the height of the disease, and the swelling of one or both cheeks is also a notable feature. This swelling is circular, hard, painful, and extensive. On inspecting the mouth, there are seen more or less extensive yellow or yellowish-grey deposits, variously prominent, on the mucous membrane of the palate, tongue, or cheeks. When opening the mouth is too painful, then the fingers can detect the existence of the pseudo-membranes on the cheeks, at the part corresponding to the external swelling. The presence of micrococci is diagnostic of the disease.

When the disease affects the trachea and lungs, cough is present; when the intestinal tract, then there is diarrhœa, fæces containing micrococci. The seat of the malady is primarily in the cavity of the mouth and in the nasal chambers; when the lungs are affected, this is probably due to inspired particles of matter from these cavities; and when the intestines are implicated, it may be suspected that portions of the exudate in the mouth have been swallowed.

The mucous membrane is not alone involved, for, in Dammann's first case, the palate bones were perforated, and a communication established between the mouth and nasal cavity. I believe the cavities of the nose are rarely involved in the diphtheritic process in man. Nevertheless, Nassiloff mentions some instances he observed in children.*

The contagium is both fixed and volatile, according to the observations made at Ludwigsburg, where the cases appeared at some distance from each other, as well as when animals were in actual contact. It appeared to be also very resisting to external influences, for when the sheds were occupied by fresh calves three-quarters of a year after they had been cleared out, these became affected in turn. Only very young

* Virchow's *Archiv*, Band 1, p. 550.

calves were attacked—their ages ranging from a few days to some weeks; cows were exempt during the entire outbreak. This exemption was attributed to the greater thickness of the epithelial covering and density of their mucous membranes, which in young animals are softer and more vascular.

The period of illness was from three to five days, and without active and timely treatment all those attacked perished.

Dammann placed a piece of diphtheritic deposit in the mouth and nostril of a four-days-old lamb. It was ill two days afterwards, and died on the fourth day. The lesions were those noticed in the diphtheritic calves. Another lamb had a piece of diphtheritic mass from a calf placed in its mouth. It remained unaffected. A year-old rabbit was inoculated in the muscular part of the thigh, and died the following day. There was a foul-looking greyish deposit around the seat of inoculation, the fascia was of a greenish-grey colour, and for some distance around the wound there were hæmorrhagic patches in the muscular tissue. Micrococci were found in the blood. Another older rabbit was inoculated in the same place as the last, with a small portion of the diphtheritic mass from a calf's tongue. It also died next day, and the lesions were similar. An incision was made in the conjunctiva of the right upper lid of the eye of a one-year-old lamb, and a morsel, about the size of a pea, of the diphtheritic deposit from the tongue of the calf No. 1 was inserted. In two days the eyelid was very much swollen, and the conjunctiva red and turgid, with a discharge from the eye and covering the cornea, as well as a yellow patch on the membrane, some distance from the point of inoculation. There was fever and loss of appetite. On the following day the inoculated mass had increased to the size of a bean, and the next day it was still larger, and the entire cornea was opaque, yellowish-green in hue, and where the yellow patch first appeared the part was very prominent, and covered with unhealthy pus. Eight days after inoculation, the cornea was removed. It was very much thickened, and when examined microscopically was found to be infiltrated not only with pus corpuscles, but throughout with myraids of micrococci. Another lamb was inoculated in the same region, but the effects were more severe.

These experiments proved, in the first place, that not only was diphtheritis of the calf transmissible to lambs, by simply placing a small portion of exudate in the mouth, and without abrading or wounding the mucous membrane, but they also, in the second place, demonstrated the extraordinary similarity,

if not absolute identity, between this diphtheritis of calves and that of mankind, the specific processes following inoculation, and observable in the conjunctivæ and corneæ of the lambs, no less than in the muscular tissue of the rabbits, being in no perceptible manner different from those noted in inoculations from human diphtheria. The rapidity with which infection takes place is also well shown in the case of the rabbits, death occurring in twenty-four and twenty-five hours.

The most important part of the narrative is yet to be alluded to. At the suggestion of Dammann, the veterinary inspector of Ludwigsburg commenced on April 29th to treat the diseased calves by dressing their mouths with salicylic acid. On May 1st, in the evening, he experienced a severe pricking sensation in the throat; nevertheless, the treatment of the calves was carried out most conscientiously. On May 5th he had to stay at home, and, when Dammann saw him on the 9th, he was in bed, very feverish, suffering from pain and dryness in the throat, great pain in swallowing, weakness of the limbs, pain and heaviness of the head, and thirst.

The cervical and submaxillary glands were swollen and tender, and the mouth was opened with difficulty. The mucous membrane was of a dark-red colour, and the soft palate and one of the tonsils were very tumefied, and appeared to be covered by a greyish-green membrane. On this day surgical and medical treatment was adopted, and on the days following, pieces of greyish-white membrane were detached by the brush employed to dress the fauces. The local disease was under treatment for ten days, but the general weakness continued until the end of June.

About the same time that the inspector was suffering from treating the sick calves, the dairymaid who attended to them was also infected, though not so severely, as she had not to go to bed. The symptoms were the same. Dammann also learned that besides these persons who had been in more or less close contact with sick calves, others in this locality had been suffering from sore throats.

Dammann himself, on May 8th, two days after he had examined the carcase of his first diphtheritic calf, had a pricking sensation in his throat; and his assistant, after dressing the mouth of one of the calves from May 11th to the 14th, had also this symptom, as well as pain on attempting to swallow. Salicylic acid gargles, at once resorted to, and continued for some time, caused the symptoms to subside and finally disappear.

As to the origin of the outbreak, nothing could be discovered, and the question as to whether it was due to transmission from mankind remained undecided. Certain it is that, at the commencement of the outbreak among the calves at Ludwigsburg, in the winter of 1874-75, the child of a coachman died of diphtheria.

I have dwelt thus lengthily on Dammann's report, as it is most carefully drawn up, and more especially as he is considered a very reliable authority in Germany; and also because the relationship of the bovine species to mankind is so intimate.

Nothing more definite is known with regard to the transmissibility of diphtheria from bovine animals to man. But of the frequent existence of the disease in these creatures there is ample evidence, and there is even some casual proof that this communicability may be a more common occurrence than is generally suspected. A recent French writer, M. Lenglen of Arras,* describes an exactly similar malady to that investigated by Dammann, but which he (Lenglen) designates as gangrene of the mouth (*gangrene de la bouche*). He witnessed it chiefly among calves, though he admits it is not special to the bovine family, as he had observed two cases of it in foals—one fifteen days old, the other about five weeks, and in lambs from fifteen days to six weeks. He did not think it was contagious, though he prescribes sanitary measures as if it were so.

Mr. Macgillivray, veterinary surgeon, Banff, also describes the same disorder, under the name of "tubercular stomatitis",† and which he asserts has been very common in his practice for fifteen years. Though it chiefly occurred in calves, yet he had not unfrequently met with it in one-year-old cattle, once in a cow, and once in a horse. The symptoms were, at first, profuse salivation, very fœtid breath, and difficulty in mastication. On inspection of the mouth, the lesions were observed inside the cheeks, about the tongue, gums, and the pharynx, or, very rarely, about the entrance to the larynx. He describes the malady as consisting in a cheesy deposit in, or degeneration of, these parts. When in the buccal region, this deposit is generally greyish in colour externally, yellow internally. He is of opinion that it is highly contagious. He gives strong evidence in favour of this contagiousness, and the description of his cases is very interesting, as showing their identity with those recorded by Dammann. But I believe he erred in considering them as of a tubercular

* *Recueil de Méd. Vétérinaire*, 1880, pp. 969, 1091.

† *Veterinary Journal*, January 1881, p. 1; June 1881, p. 377.

nature, for there is evidently nothing analogous to tuberculosis in the lesions; and, besides, calves are extremely rarely affected with tuberculosis, while sheep and horses (which he asserts he has seen attacked by the disease) are almost entirely exempt from it. The malady was evidently diphtheritis, as described by Dammann; and, in discussing its etiology, he makes some remarks which are extremely pertinent to the purport of this paper. He says, referring to one outbreak, "An interesting point arises in reference to the origin of the disease, namely, as to whether it was inherent in the first calf and communicated by it to the others, or were the whole six contaminated by the milk from the two tuberculous cows? . . . . If the disease was communicated to the calves by the milk, the question presents itself as to whether the use of the same sort of milk may not account for the occurrence of certain cases of sore throat or mouth in the human consumer. *Isolated cases of sore throat often occur among farm servants*, where ugly-looking yellowish spots or deposits are found in various parts of the throat, and even the mouth, requiring a good deal of brushing and dusting with very strong caustics. . . . . That the tubercular stomatitis of calves is contagious, is amply demonstrated in this communication; for whether we consider it as passed from calf to calf in the cases chronicled, or as conveyed to all by means of the milk, the fact still remains that it is transmissible. For my own part, I firmly believe that one calf conveys it to the other, not only by actual contact, but also if the calves are sucking the same cow, or getting their milk out of the same dairy utensils, as often happens. . . . . Whether, therefore, the contagion comes from the milk originally, or from calf to calf, if it gain access to the mucous membrane of cheeks, gums, tongue or throat, it produces in a more or less aggravated degree the tubercular stomatitis," etc.

A most serious question here arises as to the relationship of this disease of calves and human diphtheria. If the disease in the calves is produced by the milk of the cow, why may not this milk also produce the same malady in man? There is nothing at all improbable in this, looking at Dammann's experience and Macgillivray's reasonable suppositions. On one notable occasion—that of a somewhat recent outbreak of diphtheria, in the human species, at Kilburn, London—it was suggested that the malady most probably owed its origin to the existence of what is commonly known among cowkeepers as "garget", to veterinary surgeons as "mammitis", or "mastitis", among cows in a certain dairy,

from which milk had been distributed; and the question arose as to whether there was any possibility of such an origin for diphtheria. If the disease could be originated and transmitted in such a manner, then a most important discovery would have been made with regard to at least one source of a scourge, the origin of which is involved in mystery.

Is there any disease of the mammæ, or udder of the cow, which will, through the medium of the milk, produce diphtheria in mankind? At present we are without evidence to this effect. If we regard diphtheria as a specific disease, due to or characterised by the presence of certain microscopical organisms, as has now been well established, then we must trace the origin of outbreaks of the disorder to a diphtheritic source. I know of no malady specially affecting the lacteal apparatus of the cow which is of the nature of diphtheria. The disease, as we have seen, attacks young calves, very rarely cows, and is, if not at first, then very soon after, a general disorder. The question may be asked, Where do the calves receive diphtheria from? And to this we cannot as yet give a satisfactory reply. As milk is their only food at this early period of their lives, then it would seem probable that the disorder is conveyed to them through this medium, and that this fluid has been contaminated by being drawn from a diseased gland. But, as I have said, we have no evidence of the existence of diphtheritic inflammation in the udder of the cow, though there is no reason why such a condition might not be present on rare occasions—for frequent they cannot be, else they must have been noticed.

There are three kinds of mammitis—catarrhal, interstitial, and erysipelatous. But a diphtheritic kind has not, to my knowledge, been yet distinguished. Mammitis, as we know it, is most frequent when the mammary glands are active, and particularly soon after parturition; it may be due to the most varied causes, as injuries, influence of cold or variable temperature, overstocking the gland, or allowing the milk to remain too long in it, improper food—anything, in fact, which disturbs the functions of a very vascular and active secretory gland.

But there has not unfrequently been observed a form of mammitis of a phlegmonous or erysipelatous nature, which has assumed an epizoötic or enzoötic character, affecting large numbers of cows in a district, and causing serious loss, so far as the production of milk is concerned. There is every likelihood that this form is contagious, and that the milk is

dangerous as food. I am not aware that it has been observed in this country, but it has been reported upon by most excellent veterinary authorities in Germany. For instance, Professor Zurn, of Leipzig, relates* that in the summer of 1874, in a certain district, six or eight cows were affected, while grazing, with a disease of the udder. Isolated cases occurred during the following winter, and summer of 1875, but in the winter of 1875-6 the malady again showed itself to an alarming extent, no fewer than forty cows being attacked out a herd of 180.

The animals were sometimes ill for several days before the prominent symptoms of the disease were noticed. These were—the presence of a watery secretion from the udder containing coagulated masses, which gave rise to much pain as they passed through the canal of the teat; sometimes the secretion looked like white or yellow pus; the gland, as a rule, remained soft, and the temperature was seldom raised. One or more quarters of the gland were involved, and one attack did not protect from another. In many cases the part of the gland affected was totally destroyed. Dry cows (*i.e.*, cows not in milk) were attacked, as well as those in which the secretion was abundant. Suckling ewes and sows were infected from the cows, after lying or rolling about on the litter and dung from the cowsheds in which the diseased cattle stood; while the milk from these killed the calves, and that of the ewes and swine the young lambs and pigs. The samples of milk examined by Zurn did not all present the same appearances, though all were acid. When allowed to stand for some time, a greyish-white sediment settled at the bottom of the vessel, while the supernatant fluid was of a citron or dark-yellow colour, seldom of a light-green tint. After a time a very thin layer of cream formed on it. The sediment contained a large quantity of epithelium and pus-cells, and a great number of motile micrococci, which in two or three days lost their movement, clustering together and forming masses of zooglea. When the fresh milk was examined numbers of intertwined rod bacteria, forming a kind of feltwork, were noticed. Besides these, there were many cells from ·004 to ·005 millimètre long, and ·002 to ·0025 millimètre broad.

In all the cultivations made by Zurn, and which extended over a year, there never appeared any fungus except thin flocculi, composed of chains of *Torula*, resembling the yeast fungus. In other specimens the *Mucor mucedo* spores were found, which grew extensively when the milk was left for any time.

* *Deutsche Zeitschrift für Thiermedicin*, 1877, p. 381.

Zurn attributed the outbreak and extension of the malady to the micrococci and rods observed in the milk.

A similar disease has been reported upon as existing among cows, by Professor Dieckerhoff,* of the Berlin Veterinary School, who designated it "Infectious Mammitis". It was due to local infection, and to no other cause, so far as could be traced; it commenced at the extremity of the teat, gradually extending into the milk sinuses and interior of the gland. The malady was conveyed from one farm to another by persons who handled the cows.

Professor Franck, of the Munich Veterinary School, had previously drawn attention to this infectious mammitis in the cow, which he discovered to be due to bacteria.† It could readily be produced experimentally. The readiness with which it could be transmitted was doubtless due to the peculiar structure of the teat and arrangement of the milk sinuses and ducts in ruminants, as well as to the habits of these animals.

Rivolta, Veterinary Professor in the University of Pisa, also describes an infectious mammitis in sheep, special or specific in its character, a kind of septic disease.‡ But we have no mention of diphtheritis affecting the udder; though there is no reason why the mucous membrane of that gland should not be involved as well as other mucous membranes. Indeed, there is no difficulty in understanding that a calf suffering from diphtheria, and applying its mouth to the teats of the mammæ might readily infect the gland, and through it the cow. Granted, therefore, that the malady is readily transmissible from bovine to bovine and that the calf —leaving out of sight the manner in which it may become infected—may transmit the infection to the udder of the cow, there is every reason to assume that the secretion of the gland is rendered infective, and will convey diphtheria to other creatures which consume it; and as mankind can receive the disease from the bovine and other species, there is scarcely any doubt that the disorder might become spread among people, and especially young children, by milk containing the diphtheritic germs which have been developed in the udder of the cow.

That the diphtheria of animals is always derived from an infectious source may be assumed as a fact established by scientific research; that it is very similar to, if not identical

* *Wochenschrift für Thierheilkunde und Viehzucht*, 1878; *Veterinary Journal*, 1878, p. 335.

† *Handbuch der Thierarzlichen Geburtshülfe.*

‡ *Giornale di'Anatomia, etc., degli Animali*, 1875; Fleming's *Veterinary Obstetrics*, p. 695.

with that of man, is evident; that it is transmissible from animal to animal, and from man to animals, we have accidental and experimental evidence to prove; and that it may be conveyed from animals to the human species, is also a fact that is supported by the instances recorded of accidental transmission.

It must be confessed, however, that there is much yet to be learned with regard to the disease, not only in animals but also in man; and it is now mainly to experimental pathology that we must look for a solution of the obscure points, which are more particularly related to the mode in which the malady originates, and the circumstances which favour its extension as an epidemic or epizoötic scourge.*

* Since the above paper was written, the following interesting account of an outbreak of the disease, published in the *Sanitary Record* for March 15th, 1881, by Dr. McEwen, has been brought to my notice. As it bears upon the subject of this paper, I beg that it may be received as a supplement thereto. Dr. McEwen writes :—

"This painfully interesting disease,—at least, painfully interesting to those who have seen it in its most virulent form, cutting off whole families in periods of time varying from a few days to a few weeks, bids fair now to be better understood than it ever has been. It is to be hoped that, with clearer views of the etiology of the disease, the treatment may be more successful than in a great many instances it has hitherto been, and that in a short time medical men shall be saved the pain of seeing strong, healthy subjects suddenly cut off by a disease that in its worst form has so fatal a tendency.

"It fell to my lot to observe a peculiarly interesting, and fatal, outbreak of diphtheria in a rural district in the north of Scotland in the autumn of 1878. The burden of the outbreak fell on a brother country practitioner. It was in the month of August that the disease broke out at a farmhouse in the county of Aberdeenshire. Most of the individuals about the farm (including the servants) were adults. Within a short time, of nine individuals who were attacked, seven died. My friend, the medical practitioner in attendance, at this time delivered the wife of a mechanic who lived at least five miles from the seat of the outbreak. Within a few days of her delivery, diphtheria appeared in the family, and in a few days more the father and five children were dead of diphtheria. About the same time he also attended in her confinement the wife of a farm labourer in a small village further away from the seat of the outbreak. Here, too, not many days afterwards, diphtheria attacked the family, and carried off father, child, and mother. I here assisted my brother practitioner to perform tracheotomy, in the hope of saving the man's life. While in the house the wife complained of being ill and having a sore throat, and on looking into it there were diphtheria patches to be seen. I learned afterwards that she also had diphtheritic patches on the vulva. I believe there was also another instance of the unfortunate coincidence of the disease following so fatally in the wake of the obstetrical track of the practitioner in attendance at the seat of the outbreak. This "unfortunate coincidence", as I called it at the time in some notes on the treatment of some of my own cases contributed to the *Practitioner*, seems to me now more forcibly than it did even then to be more than a coincidence, and I strongly incline to the view that the public took at the time, viz., that the doctor was the medium through which the infection was carried from the original seat of the outbreak. Nothing satisfactory as to the origin of the outbreak itself could be learned, though there was a popular

notion, held by a number of people in the neighbourhood, *that the water used at the farmhouse where the disease first appeared had been contaminated by the decomposed body of a calf said to have died of some throat disease, a considerable time before, and to have been buried in such a position that the moisture from it might contaminate the water in the well from whence the drinking-water was drawn.* Popular notions are apt to be despised, and in this particular case I know the notion was despised by those whose opinions were, or ought to have been, of most value. Nevertheless, it is quite possible that the particular organism which at first caused the outbreak of diphtheria, was here generated, according to the popular view held at the time, and also that, owing to an innocent want of due caution on the part of the medical attendant, these germs were by him carried far away to other homes, there to develop. Personally I had these suspicions at the time, and I took every precaution, by washing my hands carbolically after seeing a patient with a sore throat of any kind, and by never attending a midwifery case in clothes that I had on when seeing a case of diphtheria."

REPORT ON "DENGUE" FEVER.

By J. MACKIE, M.D., Medical Officer to the British Consulate, Alexandria.

[*Read: November 29th*, 1880.]

THE disease which has prevailed in an epidemic form throughout Egypt for the last three months, and universally known here under the name of "Dengue", is supposed to be the same disease which has prevailed in other countries, East and West Indies and America, etc., and been described under a variety of names; "dengue", "dengue fever", "dandy fever", "breakbone fever", "three days fever", "scarlatina rheumatica", "aboo rukub", etc. The present epidemic outbreak may be said to be a continuation of its first appearance in Egypt, in 1877, when it broke out with great violence in Ismailia on the Suez Canal and attacked nearly the whole population of the town to such an extent, that the tribunals and commercial offices had to be temporarily closed. This outbreak continued in Ismailia from August to the end of November, and was confined to that town. It appeared again, in a mild form, in Ismailia in the autumn of 1878 and 1879, and to a very slight extent in the autumn of the present year; but, until the present outbreak of last autumn, the disease had confined itself entirely to the town of Ismailia, and prevailed only during the autumn months.

The present outbreak of the epidemic, which dates from last August, when it was first reported from Damanhour, has been severe and extensive. The disease has invaded nearly every town and village of Egypt, from Alexandria to far into Upper Egypt. In Cairo, it is calculated that nearly four-fifths of the population have suffered. It appeared in Alexandria in the beginning of October, where I had an opportunity of seeing it, and the following is a description of the disease as observed by Dr. Murison, my assistant, and myself.

It may be characterized as an "eruptive fever", having as special features great increase of temperature from the very onset, severe pains in the head and back, and in muscles and joints of the extremities. Appearance of a purplish or red

scarlatina rash, generally about the third day, but the date of the appearance of the rash is very irregular.

The disease sometimes commences with slight pains and malaise for a day or two, but, most frequently, in severe cases, commences suddenly with slight shivering, soon followed by high fever, severe pain in the head, sometimes frontal headache only, but more often severe pains all over the head, pain behind the eyes, slight redness and suffusion of eyes, and smarting pain of conjunctiva; severe pain all over the body, but chiefly in the back, and in the muscles and joints of the limbs. Severe pain is sometimes complained of at the epigastrium and in the abdomen. This is accompanied or soon followed by very high fever, the temperature soon mounting up to 104° and 105° Fahrenheit. The pulse is, generally, full and strong, ranging from 94 to 120 and 130, but in most cases I have observed that the pulse is slow, considering the high temperature, it being no uncommon thing to have a quiet, soft pulse of 84 or 90, with a temperature of 104°. The pain in severe cases is generally described as being very excruciating, and something *sui generis*—not the pain of acute rheumatism, nor neuralgia—but pain with a burning sensation, as if a red hot iron were being pushed into the joints. The tongue is generally covered with a thick, yellowish-white fur, but in mild cases is often clean. Bowels generally are constipated; nausea is often complained of, but vomiting rare. Urine high coloured, not scanty, occasionally phosphatic, sometimes more or less albuminous for a short time; and, in rare cases, slightly bloody. Skin is very hot and dry, but in some cases there are outbursts of perspiration. The face is very red and flushed, giving it a swollen appearance. In some cases, pain in the throat has been complained of, but on examination, I have found nothing but great dryness and redness from congestion.

In cases of ordinary severity the fever declines about the third day, the temperature falling to 100°-101°, and a rash sometimes resembling measles, but much more frequently the rash of scarlatina, appears in the neck, upper part of chest, points of elbows, knees and ankles, but often covering the whole body, especially in children, where I have observed it to be generally of the character of measles. The rash sometimes appears from the commencement; but more often about the third or fourth day, and not unfrequently much later, after patients have considered themselves convalescent and resumed work. The duration of the rash is indefinite, generally disappearing in four or five days, but often lasting much longer. Generally there is no desquamation after the

eruption fades—desquamation properly so called, as occurs after scarlatina—but in some cases where the rash has been bright red and well-developed, it is followed by a fine furfuraceous scaling, especially on hands and arms. Convalescence after a severe attack is very slow, patients remain weak and prostrate, requiring liberal administration of nourishment and stimulants, beef-tea and port-wine, etc. Sleeplessness is also a common and troublesome symptom during convalescence, if the attack has been severe. Giddiness and temporary impairment of memory have also been noticed in some cases during convalescence.

Children as well as adults are liable to be attacked; but as far as my observation goes, children suffer less severely, and in them the rash has generally been of the type of measles; but sometimes also bright red. They do not suffer from protracted convalescence nor great prostration. They soon regain their "looks" and spirits. I have had under my care children as young as six weeks and three months, with temperature of 103-5°, and well-developed rash. I have seen no case in an adult over fifty-six years of age. In children the disease is sometimes ushered in by convulsions; in adults it is sometimes accompanied by slight delirium at outset; and in hard drinkers a quiet delirium resembling mild delirium tremens is frequently observed during convalescence, the delirium amounting simply to a confusion of ideas, restlessness, seeing strange people, birds, insects, etc., in the room, but at the same time being clear enough to know, when for a moment they collect their thoughts, that the visions are not real. This soon passes off as the strength improves.

The above is the history of an ordinarily severe case of "dengue", as it has come under my observation during the present epidemic here; but many of the cases are of a much milder form, consisting of slight pains in head, pains all over the back, slight giddiness, nausea, and malaise, with a mild degree of fever. In these cases many people never remain indoors, but continue, though with difficulty, at their daily work. Many of them are to be met at out-door work, with their hands and arms, and often also their body, covered with a red rash, and feeling ill, but, as they often say, not sufficiently ill to lie up. In many of the milder cases no rash on the body appears, and is often supposed not to have existed; but my experience is, that the rash, to a greater or less degree, is almost universally to be found if carefully looked for. It may not be on the face or trunk, but may exist merely as red patches on points of elbows, knee-caps,

neck, or upper part of sternum. In one or two cases I have found enlargement of liver and spleen during the fever only, but requiring no special treatment. If any one may judge from the behaviour of the disease during the present epidemic, it would seem to be eminently contagious. Once it attacks one member of a household, the whole household almost to a certainty suffers.

The treatment found most useful by Dr. Murison and myself, and which has certainly in almost every case given almost immediate relief to the pain and lowered the temperature, has been the administration, at the commencement, of two or three half-drachm doses of salicylate of soda, at intervals of three or four hours. The pain in the head has often subsided, as if by magic, after the first or second dose. A saline purge or two after this, of some mineral water, has often been all the medicine required. During convalescence after a severe attack, a liberal allowance of wine, beef-tea, Valentine's meat-juice, etc., to combat the prostration, with good nursing and constant attention, as fainting fits were not uncommon. The sleeplessness during convalescence I have found best treated by a combination of chloral and bromide of potassium. In some cases a slight degree of fever has continued during convalescence, with great restlessness and delirium, dry tongue, etc.; but I never found it necessary to depart from the above treatment, and found that under it the symptoms soon subsided, though I am aware that some here have thought it necessary to apply ice, leeches to head, and other depletory measures. In mild cases little treatment has been found necessary. An emetic of ipecacuan, if there was much nausea; a mild purge, and perhaps a dose of salicylate of soda, if the pains were severe.

At the outbreak of the epidemic, many medical men here, regarding the disease as malarial, treated it by administration of large doses of quinine. I have found nothing in the symptoms or progress of the disease to lead me to the conclusion that it is of malarious type, and have not treated a case with quinine during the epidemic. Repeated attacks are not uncommon, some people having suffered two or three times. I have heard of no fatal cases, though some severe cases have presented rather alarming symptoms, such as temporary albuminuria and hæmaturia during the fever, and depression and prostration, attacks of syncope, vertigo, dry tongue and delirium, with restlessness and sleeplessness during convalescence. These symptoms, in the cases treated by Dr. Murison and myself, have all soon improved under

the treatment mentioned above. In one or two cases that came under my observation the patients subsequently suffered from boils of an unhealthy sloughy nature; others, who presented no peculiar symptoms during convalescence, remained weak and depressed for several weeks.

The above is a description of dengue, at present prevailing extensively in Alexandria and other towns and villages of Egypt, as it has fallen under my observation.

ON A

SYSTEM OF DETACHED FLOATING BARRACKS FOR THE FORMATION OF A SMALL-POX OR FEVER HOSPITAL.

By V. B. BARRINGTON-KENNETT, M.A., LL.M., late Chief Commissioner of the Stafford House Committee.

[*Read: June 22nd*, 1881.]

It is with feelings of diffidence, and trusting as a layman to your generous indulgence, that I have accepted the invitation of your distinguished President, Sir Joseph Fayrer, to lay before you the following scheme for a Floating Hospital, composed of a system of detached barracks constructed on the decks of barges.

My suggestions are founded upon several years' practical experience of different hospital systems, during the principal European wars since 1870, while acting as Commissioner in charge of large ambulance operations, and, as such, having the benefit of the advice of eminent medical men of various nationalities. The system here proposed is somewhat similar to one which I used successfully on the Danube for the National Society for Aid to Wounded during the Turco-Servian War 1876-77, with the assistance of Baron Mundy, the well-known authority on military hygiene. Previous experience in the Carlist War, had led me to the conclusion that hulks of old ships were not well adapted for hospital purposes, except in the case of barracks built on the decks. The ventilation below deck was generally carried out with difficulty, especially when the ship was at anchor, and it was found almost impossible to thoroughly purify the hulk.

From a sanitary point of view there can be little doubt of the great advantages resulting from the distribution of small-pox and fever patients in detached barracks; this system I propose to apply to the case of a floating hospital on the Thames.

I shall treat of the subject under the following heads. First, the construction of the individual barracks, and the system of grouping them; secondly, transport arrangements by land and river between the Metropolis and the hospital.

The barracks should be constructed on the decks of large

barges of not less than 120 to 150 tons, or, better still, on platforms resting on sets of two such barges rigidly connected, side by side, somewhat after the design of the twin ship *Castalia*. The material used should be wood or corrugated iron; the latter is more easily disinfected, but, on the other hand, is more costly; moreover, there is great difficulty in regulating the temperature of the interior, especially under a hot sun.

Every barrack should cover the entire deck or platform, with the exception of a few feet fore and aft, and a narrow passage round the bulwarks. The roof should be high and sloping, the whole exterior resembling the popular form of a Noah's ark.

The floating barracks should be arranged in groups of three; the members of every group being moored in *échelon;* thus, the bow of the second barge would be opposite to the midships of the first, and at the distance of half a barge length from it. A platform will connect the stern part of the first barge with the forepart of the second. The third should be similarly situated with regard to the second, and communicate with it in like manner. By this system of *échelon* there is secured, immediately in front of the windows, the maximum amount of fresh air and space compatible with convenient means of communication. The platforms can be used by convalescents for exercise.

Similar groups of barges might be moored lower down the river, the first of the second group being, say, at two barges' length from the last of the first group. The direction of the *échelon* must be accommodated to the extent of space in the river allotted to the hospital. The general line of barges would thus form a zigzag, the attendants' and other auxiliary barges being moored in the angular spaces left between the lines of the zigzag.

The following auxiliary barges will be desirable. 1. A barge for the accommodation of the resident staff, which can also contain kitchen and store room.—2. Laundry barge for washing and disinfecting the linen.—3. A mortuary or *post-mortem* examination barge. One of the barges of the hospital should be reserved for doubtful cases, or for cases of mistaken diagnosis. I might mention that the barges being in *échelon*, the drainage of one barge will not be carried in the direction of its immediate neighbours; at the same time, the usual precautions might be taken to disinfect everything that passes from the barges.

With regard to ventilation, much depends on the position of the windows, which should be ranged along each long side

of the barrack alternately, each window facing a blank space on the opposite wall. In the case of a small barrack, when windows exactly facing each other are opened, a draught of fresh air coming in at those on one side, is apt to pass out at the opposite ones, leaving a considerable proportion of stagnant air in the interior undisturbed. If the windows do not face each other, a portion of the fresh current of air introduced strikes the opposite wall, and the remainder is directed to the right and left towards the nearest open windows and upwards through the egress ventilators. Thus the whole current is broken up and dispersed, creating less draught and more ventilation than in the former case. The windows should open top and bottom, and it would be an advantage if the upper portion could open by slanting inwards. Every barrack should be furnished with supplementary means of ventilation, the roof being provided with exhaust cowls on the Banner or Buchan system, to assist the rapid egress of foul air.

Fresh air can be introduced by means of tubes on a system similar to Tobin's, one open end of the tube being outside the barrack, the tube being then introduced to the interior at the level of the floor, and carried vertically upwards three or four feet along the wall. The column of fresh air introduced is warmed by the atmosphere of the barrack before striking the roof and being dispersed. Should the external air be very cold, the tube can be warmed; in this, however, great nicety is required, as the air, if too much heated, will become dry and injurious. The ordinary windsail can be used with advantage. Fresh air can also be conveniently introduced by means of conical funnels, the smaller aperture being outside and the larger inside, which has the effect of dispersing the air without draught. Portholes should be made under the deck line, so as to keep up ventilation in the hold. This can be used for storage purposes, and for hot water heating apparatus.

The heating of the barrack can be best effected by means of hot water apparatus, the water being heated by a boiler in the hold. I have found it undesirable to have any stoves in the interior of the barrack itself, and any system of warming by heated air is considered injurious to patients. The hot water system is exceedingly safe and is easily regulated according to the temperature required.

I abstain from going into minute details as to the interior fittings of the barrack, which must be left to the discretion of the authorities in charge, and depend upon financial and other considerations. There should be a door in the side of the barrack, so that patients can be conveniently lifted in from

the transport barge when brought alongside. The beds should be of iron, furnished with seaweed mattrasses, and the legs should rest on small India-rubber cups, which have the effect of keeping the bed steady, especially in case of inequalities in the surface of the deck or platform.

For the convalescents and attendants, I recommend an ingenious bed invented by Mr. Squib of St. Heliers, Jersey; the bed revolves into a vertical position when not in use, and a wooden slab or locker, fixed to the bottom of the bed, is lifted up and forms a table when the bed is vertical. The construction of this bed is simple, and the cost is exceedingly small. By this means the interior of the barrack can be made very roomy and comfortable when the beds are not in use.

The transport service from the Metropolis can be carried out by means of two barges, one to contain twenty patients and the other four, to be towed by a steam launch attached exclusively to the service of the hospital. One wharf should be selected on the Thames and another on the Lea. This river, passing as it does near the densely populated districts of the East End, could be utilised, not only for the conveyance of fresh patients, but for transporting the convalescents from Homerton hospital to the floating barrack, should it be considered desirable.

The patients should be conveyed from their homes to the wharves in specially constructed ambulance waggons, fitted with India-rubber slings for the suspension of stretchers. The two transport barges should be constructed on somewhat similar principles to the other barges, but instead of having beds, should be furnished with slings for suspending stretchers, similar to those in the ambulance waggons. The stretchers will have short legs, so that they can be rested on the floor without inconveniencing the patients. These should be moved from their beds in their own homes on to the stretchers, and slung up in the ambulance waggons which will convey them to the wharves. On arrival, they should be slung up in the barges, being still in the same stretchers, and thus conveyed alongside the floating hospital. The stretchers can then be lifted into the barrack, and thus the patients will never be removed from them, or require any handling until placed in the beds they will permanently occupy.

We carried out this system successfully during the Turco-Servian War. The wounded were conveyed by road for two days, and by a hospital barge on the Danube for a third day, on the same stretchers. These were made on the design of Baron Mundy, and though very light and portable, were

almost as comfortable as a bed, to sleep on. The class of stretcher for the transport service in the Metropolis would be larger than those in general use in war, and might be furnished with a thin mattress to prevent any chill to the back of a patient.

The main object which I have in view in these last suggestions, is to prevent the handling of patients during transport more than is absolutely required, thus lessening their discomfort and diminishing the danger of infecting the attendants. It must be remembered, that the transport attendants are those who come mostly into contact with the outside world, and, therefore, this precaution is an important one. These attendants should wear a distinctive dress, and should, as long as they wear this dress, be considered infected, and should be avoided; moreover, it should be made a penal offence to allow them to enter any dwelling house, or place of public resort, except with special leave, when they should be supplied with other clothes and be themselves disinfected. They should be responsible men, specially selected for trustworthiness and intelligence, and should be well paid.

In answer to any objection which may be raised as to fatiguing a patient by the barge transport, I can testify, from personal experience, that there is no motion so easy as that of a barge being towed. The form of the barge causes a steady resistance to the rope, which is thus kept at an almost constant tension, and prevents by its elasticity the slightest feeling of vibration. The steam launch used for towing should carry a distinctive flag, and orders given that all craft on the river, should, as far as possible, give way to it. Conveyance in a barge towed by a steamer is far preferable to conveyance in the steamer itself, both on the point of avoiding vibration, and also preventing the possibility of infecting the steamer, which may be used for other purposes connected with the hospital.

The most important precautions to be taken in the case of a floating hospital, are those against communication with the shore, collision, and fire. Strict quarantine could be enforced by a small body of river police, a heavy penalty following any infringement of the regulations made. During the day, a yellow flag should be hoisted, and at night, a brilliant light displayed to warn off passing craft. Every barge should be furnished with one of Dick's well known "Extincteurs", in case of fire, and, in addition, a small hand fire engine should be kept on board one of the auxiliary barges.

Telephonic communication can be carried on between the barges, and also between some central barge and the shore,

where a small shed should be constructed for friends and others wishing to communicate with those on board. The wires should be sunk with heavy weights immediately below the barges, so that they should lie on the bottom of the river, and thus be out of danger of being broken by passing craft. Should the portion of the river between the barges and shore be much used for anchorage, there would be an objection to the wire communicating with the shore. Telephonic communication could also be conveniently established between the wharves and other stations in the Metropolis.

In the selection of a site on the Thames for a hospital, much must depend on the size of the barges, and on whether a system of twin barges or single barges is used. There are sites near the south shore of the river below Greenhithe, and near the north below Gray's Thurrocks; these are protected from the easterly winds by bends in the river, and the latter is, in addition, sheltered by rising ground towards the east. Should these sites be considered too exposed, or too far removed from the Metropolis, others could be chosen higher up.

I trust that the following advantages, or at least some of them, may be attained:—1. An easily enforced system of quarantine and isolation of the entire hospital from the main land. 2. Isolation of every group of barracks from neighbouring groups. 3. Any floating barrack can be removed to another site without difficulty, and thus, convalescents may be separated from others, and removed to another locality—perhaps, nearer the sea. 4. Any barrack, when emptied, can be towed away and thoroughly disinfected before receiving another set of patients. 5. The cost of constructing such a hospital would not be more than one on land, taking into consideration the value of the land, and deterioration of neighbouring property. In the case of the floating hospital, there could be no claims for compensation. 6. Patients from the Metropolis can be transported to the hospital in the transport barges as described, thus obviating the danger of infection in conveyance by rail, and the great fatigue of the alternative—a long journey by road. 7. The amount of accommodation in the hospital can be increased or decreased at very short notice; the barracks can be built, on barges, in a different locality from the hospital, and then floated down, thus removing all danger of infection to workmen.

I may add that the suggestions, which I have had the honour of laying before you now, were made long before the Metropolitan Asylums Board had accepted the liberal offer of the Government to place the ship *Atlas* at their disposal.

No doubt the Board has used a wise discretion in accepting that offer, and it is to be earnestly hoped that the accommodation thereby afforded, supplemented by the tent convalescent hospital, will be found sufficient. However, as I pointed out to Dr. Collie, the able medical superintendent of Homerton Fever Hospital, when he consulted me on the subject, my suggestions as to transport arrangement by land and river, systems of ventilating, warming, and telephonic communication, are equally applicable to the case of the proposed *Atlas* Hospital; while a supplementary floating barrack would always be a valuable addition to the institution.

In order to secure the efficient working of this scheme, especially as regards the transport service, combined action and harmonious co-operation on the part of the authorities of the different districts and the Metropolitan Asylums Board is absolutely necessary. The expenses of transport might be borne by every district rateably, according to the number of its patients conveyed.

Should these suggestions be approved of, I shall be happy to supply any further details as to the practical working of the scheme. For the present, I trust that what I have said may lead to a discussion on the subject among those I see around me, who are so eminently capable of judging of its merits; thus the scheme may eventually be carried to a practical issue, not only for a temporary hospital, but as a permanent institution.

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